BACILLARY CONSUMPTION

BY

DR. HORACE DOBELL.

1925

Edgar Marsh Crookshank

SAINT HILL SUSSEX.

ON

BACILLARY CONSUMPTION:

ITS NATURE AND TREATMENT IN

THE TRUE FIRST STAGE.

BY

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Intolerant extremes are but the kicks of Ignorance, to which the Wise act as buffers, to keep the balance true.

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Dedicated

TO MY ILLUSTRIOUS COADJUTOR AND FRIEND,

PROFESSOR VILLEMIN,

WHO OPENED THE GATES FOR THE DISCOVERERS

OF THE BACILLUS TUBERCULOSIS

BY HIS WISE AND BRILLIANT EXPERIMENTS ON THE

Inoculability of Tubercle.

PREFACE.

THE first draft of this treatise was written in 1887, and comprised all stages of Consumption, with tables of cases and analyses of sputum for Bacilli and *débris* of lung tissue. But the subsequent publication of many valuable contributions to these subjects—especially those of Professor Arthur E. J. Barker, Dr. A. M. Brown, Dr. Percy Kidd, Dr. R. W. Philip, Dr. Francis Troup, Dr. G. Sims Woodhead, and the Drs. Williams, in this country—determined me to completely re-arrange my work and to restrict it to a consideration of the nature and treatment of the INITIAL STAGES of Consumption.

The investigation of Tuberculosis moves on so rapidly, of late, that the accumulation of materials outstrips the pace essential to considerate composition and publication; hence several important contributions which have appeared since this work was placed in the hands of the printer could not be noticed. But so far as practicable, consistently with these inevitable conditions, I hope I have kept my facts fairly "up to date."

The italics, in my quotations from various authors, *are my own.* They are only used to emphasise the passages which especially refer to the subject under immediate discussion.

STREATE PLACE, BOURNEMOUTH,
December, 1888.

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ON

BACILLARY CONSUMPTION.

CHAPTER I.

The difficulty of the Medical Practitioner is to do the best for his Patient, without waiting for the ultimatum of Scientists—The object of this Treatise is to help him in this difficulty regarding the True First Stage of Consumption—The discovery of the Tubercle Bacillus need not destroy the value of accurate antecedent observations; but it may vitiate hypotheses—These must be reconsidered after each new discovery—The proper place must be assigned to the Bacillus, Etiologically, Pathologically, and Clinically, and its influence on Therapeutics decided—List of Questions stated, to be answered in Chapter V.—Contributions of Barker, Brown, Coats, Crookshank, Kidd, Klein, McFadyean, Philip, Troup, Watson Cheyne, Weigert, Williams, Woodhead, the Congress at Paris, and the Author—Bacilli in Milk—Description of Tubercle—Epithelial, Epithelioid, and Giant Cells.

In the commencement of the American Civil War, the North was stirred to its core by the following rough lines vociferated by a patriot whose zeal out-did his English :—

> "Here's Hell let loose, and we lie flat,
> And all the world around is singein',
> While General This and Governor That
> Are quarrelling o'er the garden engine."

A somewhat similar spirit of zealous impatience is apt to stir the mind of the medical practitioner, who is

called upon to prescribe, somehow or other, for patients threatened with death from diseases concerning which questions as to their exact nature are still seething in the cauldron of scientific discussion. He cannot wait for the ultimate results of the scientific investigation of disease! He naturally exclaims, "Tell me, in the state of our knowledge *this day*, what is the best thing I can do to repel 'this enemy at our gates'?"

The doctor is amply justified in this impatient demand, and with equal right it is reiterated by the public.

The purpose of the present treatise is to attempt some sort of articulate answer to this call with respect to "The True First Stage of Bacillary Consumption."

It has been the first object of my life-long study of Tuberculosis to discover the nature, causes, and symptoms of the pre-tubercular stage, in order to find out the means for its cure, and thus to be able to prevent the occurrence of tuberculisation.

We will proceed at once to consider the position which these questions hold to-day (see pp. 42-46).

The announcement in 1882 of the discovery by Koch and Baumgarten of the tubercle bacillus, necessitated that every physician, whether scientific, practical, or both, should reconsider his position with regard to the etiology, diagnosis, and prognosis of consumption, but especially as to its prevention and treatment.

I had just completed and published a reconsideration of more than thirty years' work on the subject,* the whole of which had preceded the discovery of the bacillus. I stated that my work had been dominated by the conviction "that intelligent, accurate, systematic

* "On Loss of Weight, Blood-splitting, and Lung Disease." Second Edition, 1880.

clinical observation is the source to which we must principally look for the discovery of the nature, cause, sequence, prevention and cure of such affections as destructive lung disease;" and in reference to "the opinion held by some that the causes of consumption are not general, but local—that they begin in the diseased organ itself not in a constitutional state"—I added that "no question in practical medicine can be more vital than this. It strikes at the root of all ideas of prevention as well as of treatment, and the only way to clear it up is to push a searching investigation into the early clinical history of destructive diseases of the lungs:—

"1. To ascertain in what proportion and at what intervals the earliest signs or symptoms of local disease are preceded by signs or symptoms of constitutional disease.

"2. To ascertain the typical characters of the signs and symptoms of such local and constitutional diseases.

"3. To ascertain whether the earliest stages of local disease can exist without indicative signs and symptoms. It is here that the aid of morbid anatomy may be called in with signal value in the class of accidental cases (referred to in the work).

4. To accumulate any cases which appear to be exceptions to general rules. To scrutinise these with the greatest care, and endeavour in every possible way to explain their exceptional character. If it is found that in a large number of cases of pulmonary consumption (destructive lung disease with constitutional decline), local disease is preceded by constitutional disease by an unequivocal interval; and yet that in a certain number of cases local disease precedes constitutional disease by an unequivocal interval; the conclusion is almost inevitable

that *there are at least two modes in which pulmonary consumption may commence.* It then becomes a point of the greatest interest and importance to ascertain whether these two classes of cases are to be considered as absolutely distinct throughout, or whether there is a period in their causative history at which they meet on common ground. That is to say, is there a stage at which the constitutional disease sets up the local disease? and is this first stage of the local disease, in these cases, the same as the first stage of the local disease in the cases *not* preceded by constitutional disease? And again, starting from this point, are the subsequent symptoms of constitutional decline in the two sets of cases *due alike* to the effects of the progress of the local disease? or are they, in the first set of cases, due to the progress of the original constitutional disease plus the effects of the local disease? And, in the second set of cases, are they due to the effects of the local disease alone? Again, does this local disease set up a state of constitutional disease of the same nature, and leading to the same local effects as that which precedes the local disease in the *first* set of cases? or is a new constitutional disease, of special character, set up by the local disease in the *second* set of cases? And, if so, is the local disease of the *first* set of cases also competent to set up a special constitutional disease the same as that of the *second* set? Finally, are the symptoms of constitutional decline which follow the establishment of local disease in *each set* of cases devoid of all special character and only such as may accompany the progress of any local disease proceeding to a fatal termination." *

Now, I think it will be admitted by all fair-minded

* See the answers to these questions, Chap. V.

men that such an inquiry as I have detailed, honestly conducted, must yield results the importance of which will not be destroyed—although they may be modified—by any new discoveries of facts; but that, on the contrary, the results of such an inquiry will aid us in associating newly discovered facts with appropriate ideas as to their place, sequence, and import. This is strikingly illustrated by the case of the discovery of the bacillus which followed close upon the heels of the publication of my answers to the foregoing questions. It will be found that it readily takes the place of *one of the irritants* enumerated by me as causes of tuberculisation, the one which *then* I could only hypothetically explain (see pp. 42, 62). It also readily takes the place of "the virus," the existence of which was asserted as a general conclusion from "the discussion on the Pathology of Phthisis Pulmonalis" at the Pathological and Clinical Society of Glasgow, February and March, 1881, just after the publication of my work, but still before the discovery of the bacillus.

Dr. Joseph Coats said, "We cannot accept any theory of phthisis which does not take into account a state of the system predisposing to it. . . . That a state of the system is not sufficient to account for tuberculosis is abundantly evident in cases of acute general tuberculosis. I have seen a strong muscular man with no lack of adipose tissue die within a few weeks of this disease, evidently a *virus* of the most active kind. . . Physicians are, perhaps, liable to take too much the appearances during life into account. And in this reference I would say that pathology undoubtedly points to a *virus* as the cause of phthisis, and clinical facts point to a state

of the system as at the bottom of it. We have to accept both, and my belief is that this position will be that of the immediate future."

As these were the inevitable conclusions from collected observations—so far, of course, as those observations had been accurately made, and the conclusions rationally drawn—they could not be vitiated by the discovery of the bacillus, that is, of a fact which, although not known, yet existed, and exerted as much influence upon the clinical characteristics of the cases, while unknown, as it could exert after its discovery. Therefore, such clinical observations of facts must still take their place among the foundations of sound medical and scientific conclusions.

It is quite different with regard to any theories or hypotheses based upon them. These must inevitably require reconsideration whenever a new discovery of facts is made. And no discovery could more emphatically call for such a reconsideration than that of the Tubercle-Bacillus. Therefore, the moment its discovery and the means of its identification became known to me, I instituted a new series of observations with a view to endeavouring to estimate its clinical importance, and to assign it its appropriate place and sequence in relation to my former observations. From that time to the present the sputum of all my chest cases has been submitted to a double analysis, first for bacilli, second for débris of air-cells (by Fenwick's process); and I have done my best to keep *en rapport* with the work which has been prosecuted in all parts of the civilized globe. No praise can be too great for the spirit and untiring industry with which the rising young physicians and scientists have applied themselves to this subject, both in the laboratory

and at the bedside. A mass of accurate observations has been accumulated, which is astonishing when we consider that it was only in 1882 that the discovery of the tubercle-bacillus was announced.

This ghastly denizen of our microscopic world, although so recently unveiled, is no new comer. We find that our forefathers pickled it unconsciously, and put it away in their bottled specimens of "Tubercle" to be found in our museums. It was as busy at its secret work then as now; and, therefore, as I have said, all clinical and pathological observations truthfully made and recorded, in whatever day this was done, relate as much to the work of the bacillus as do those made yesterday—so far as the means of observation and record of each day have been equal to the task.

It is the duty of the investigators of to-day, as it will be the duty of those yet to come, and as it has been the duty of the investigators of the past, truthfully, circumspectly, candidly, and I will add reverently, to seek to find out the exact place which each newly discovered element has hitherto secretly filled in the great histogenetic puzzle of metabolism.

We are all too apt, especially in the fervour of youth, to think that each newly-found wonder must upset all that went before! But the more sedate wisdom of experience teaches us that this is not so—fortunately indeed that it is not so, for otherwise the foundations of human knowledge would be as shifting as the sands of the sea. The plan of the great puzzle is the same, though each new cycle of scrutinizers may find that its elements are again and again divisible.

The question now before us is no longer whether there is a specific bacillus tuberculosis? but what is the place

which it now occupies and always has occupied in the histogenesis and metabolism of tuberculosis?

It is fortunate for my present purpose that, recently, several important contributions have been made to the clinical and experimental history of the tubercle-bacillus and its effects, by men having at their command exceptional opportunities of research in laboratories, museums, and hospitals. I especially refer to the works of Barker, Brown, Coats, Crookshank, Kidd, Klein, McFadyean, Philip, Troup, Watson Cheyne, Weigert, Williams, Woodhead, and the reports of the Congress on Tuberculosis which met at Paris in July, 1888. These bring our knowledge quite "up to date," and thus completely fill the important interval since the publication of my Resumé in 1880, and the discussion at Glasgow in 1881 which occurred immediately before the discovery of the tubercle-bacillus, gave a *point d'appui* to the surmises as to the nature of the specific inoculable "irritant" or "virus," the existence of which most investigators had ceased to doubt.

Mr. Barker,* after enumerating the leading facts which have been gradually accumulated in support of the view that scrofula and tuberculosis are identical, says: "But though all these facts seemed to point very clearly to the identity of scrofula and tuberculosis, it was not until the discovery of the bacillus tuberculosis, and its demonstration in the initial lesions of the two affections, that positive proof was actually forthcoming; almost at the same time Koch and Baumgarten pointed out that in typical tubercular and scrofulous nodules

* "Tubercular Joint Disease, etc. Lectures delivered at the Royal College of Surgeons, England, June, 1888. By Arthur E. J. Barker, F.R.C.S." Reported in the *British Medical Journal.*

this organism could invariably be demonstrated by different methods of preparation; . . . and it is a noteworthy fact that many of those who at first, on the strength of their own observations, had stood out most firmly against the theory of the specific or parasitic nature of tubercle, have since become its most ardent supporters; and, moreover, have given in their adhesion to the view that this bacillus is the specific organism of tuberculosis. . . . Experimental research repeated, with proper precautions against error, left no doubt on the subject, and not only was the inoculation of fresh tubercle shown to produce in all cases a definite tuberculosis, but it was also demonstrated that the organisms in question, *cultivated for many generations* in various media, until all trace of contamination with matters from the original living tissues had been got rid of, were capable of producing this disease, when introduced into the animal body, with as great a certainty as deep sleep is produced by the hypodermic injection of morphine. Again, the bacillus could be obtained in any quantity from tubercles produced in internal organs by the inoculation of these cultivations in distant parts, and could be multiplied *ad infinitum* by cultivation, and propagated among some of the domestic animals with unerring certainty. Now, it mattered little whether the tissue from which the inoculations were made was taken from a scrofulous joint, or from a typically tuberculous lung, the result was practically the same. . . . The researches bearing upon these facts are far too numerous for detailed description . . . But to a candid mind they bear but one construction, viz., that tuberculosis and scrofula are diseases produced by the introduction into the animal body of a specific poison, and that this

poison is always associated with the presence of the bacillus in question, if it be not actually the organism itself, and further, that without the presence of this organism typical tubercle does not exist."

Dr. Troup * sums up the matter by saying, "The only thing certain, however, is that the lung disease is tuberculous where they (the bacilli) are present."

Dr. Sims Woodhead † says, "There is now, however, sufficient evidence to justify pathologists in stating that many of those forms which different clinical observers have from time to time described as tuberculous are undoubtedly tubercular [bacillary ?] in character; from the grey, gelatinous, or fibroid nodule, to the large caseous masses, leading to cavity formation; and the presence of the specific bacillus has time after time been demonstrated in all these forms, both by staining and by inoculation. There can be little doubt that these forms are essentially the same, and that the differences observed are due firstly to the *resisting power* of the tissue attacked, and secondly, to the *numbers and activity* of the attacking bacilli. If the behaviour of other tissues under the action of mechanical or micro-organismal irritants be borne in mind, there will be little cause for wonder that there should be these numerous varieties of manifestation of the action of the specific irritant in tuberculous lungs."

We cannot attach too much importance to the fact stated by Mr. Barker, that the bacillus is still the

* "The Diagnosis of Early Phthisis by the Microscope. By Francis Troup, M.D." *Edinburgh Medical Journal*, July, 1888.

† "Tuberculosis and Tabes Mesenterica. Lectures delivered before the Honourable the Grocers' Company in the University of London, July, 1888. By G. Sims Woodhead, M.D., etc." (Condensed report in *The Lancet*.)

specific infective element of inoculable tubercle after having been cultivated, for many generations, in various media till all traces of contamination with matters from the original living tissues have been got rid of; because this disposes of the suspicion, which naturally arose when the bacillus was first discovered in tubercular matter, that it might not be the bacillus itself, but some matter which it carried with it which was, in truth, the specific inoculable element.

After the deepest thought and elaborate reconsideration of the whole subject, in the light of my former investigations and of all that has happened since, I accept as a fact that the bacillus tuberculosis—the organism itself pure and simple—is the specific transferable element, capable, under certain conditions, of domiciliation in animals, and the presence of which in a tissue is capable of setting up the formation of tubercle (see pp. 29, 43, 54). But to *call the bacillus "tubercle" would be absurd*, for it is not proved to be more than one of its causes. Tubercle, however produced, is more or less of what I described, in 1880,* as "a foreign mass made up of hyperplasia, of adenoid tissue, epithelial and connective tissue proliferation, angio-plasia, and other attempts at repair of disintegrated tissues, and the results of degradation and decomposition of these new formations." In this description "angioplasia" was made to include "*giant cells.*" It is suspected by many that giant cells may have a specific relationship to the bacillus, and should this prove to be true, they ought not to be present in any mass, not caused by the bacillus. Their presence in that case would be as typical of disease caused by the bacillus as the presence of the bacillus itself; although, if from

* "On Loss of Weight," etc. (*op. cit.*) Second Edition, p. 170.

any cause the bacillus had disappeared from the mass its former presence had set up, the mass would no longer be capable of transferring the disease by inoculation It would cease to be infective.

In this way it might puzzle experimenters, and lead to confusion in the conclusions drawn from their experiments. It is of the utmost importance, therefore, to bear in mind this possible source of fallacy, and to continue our researches into the life-history of the giant cell, and of the other essential elements of the mass we call tubercle till absolute certainty is arrived at.

Klein says,* "In human tuberculosis, in tuberculosis of cattle, and in artificially induced tuberculosis of guinea-pigs and rabbits, there are met with tubercles in various stages—young and adult—in which no trace of a bacillus is found; whereas in the same section caseous tubercles may be present containing numbers of tubercle-bacilli." (See p. 43.)

Dr. Woodhead † and Professor McFadyean carried out a most careful and systematic examination of over six hundred cows in the Edinburgh dairies, and they "found thirty-seven beasts in which there was mammitis, but only six (16 per cent.) in *the milk* of which they could demonstrate the presence of tubercle-bacilli, and then only in small numbers. In one of the six, and subsequently in five other cases, they made sure of the existence of the bacilli in enormous numbers in *the udder* by microscopic examination. They found new tubercular tissue disseminated in patches of various sizes throughout a portion of the gland, and that all the more minute elements of

* "Micro-Organisms and Disease." Third Edition. By E. Klein, M.D., F.R.S., etc., 1886.

† "Lectures," *op. cit.*

tubercle could be distinguished—the small round cells in which the nuclei are comparatively large, and the epithelioid cells, between or amongst which is a fairly well-developed reticulum. The giant cells were very numerous, but not so well formed as in the human subject; they were scattered throughout the new tissue. The tendency to caseation of tubercle in the udder is not nearly so well marked as in other parts of the body, but it does undoubtedly occur at points. The new growth of tuberculous tissue gradually invades the lobules of the gland, passing in along the lines of the lymphatics of the interlobular septa, so that a gradual transition from the healthy gland substance to the dense tubercular mass may be seen. In the mass itself the characteristic bacilli are present in almost inconceivable numbers. They are seen first as small stained rings (masses of bacilli) around a slightly granular or homogeneous mass—in fact, the giant cells seem to consist of *the débris of cells, the result of the activity of the bacilli.* In the smaller cells bacilli may also be seen, and others may be demonstrated lying in the spaces between the cells. On careful examination of the more healthy parts of the gland, especially at the margin of the new growth, ulceration into the ducts may be made out. In consequence of this interference with the nutrition of the tissues immediately around the ducts or acini, the basement membrane has given way, and a mass of tubercular granulation may be seen projecting into the lumen ; the epithelium is also proliferating. In the granulation tissue, in the epithelial cells, and even lying free in the lumen, there are frequently numerous bacilli, and it can be easily understood how, once in this position, they find their way into the milk. This ulceration is not, however, of such

frequent occurrence as might be expected, for in the greater part of the gland substance left there is little or no catarrhal proliferation, and the ducts and acini appear to be *obliterated in great measure by compression.*" Dr. Woodhead pointed out that, "in connection with this, it would suggest itself to most of those who examined carefully the sections he exhibited that what are called giant cells may in reality be nothing more than acini or ducts in which the bacilli have attacked and destroyed the epithelium. This idea would be greatly strengthened if some of the larger ducts were examined, for in these similar degenerative changes of the epithelium could be seen; in this epithelium bacilli could be demonstrated, and in the lumen itself there are frequently similar granular or homogeneous masses in which a few bacilli may be seen. In addition to these positions bacilli could be demonstrated in epithelial cells still attached, and also, in rare cases, in those lying free in the apparently healthy milk ducts, in which position Professor McFadyean had first demonstrated them." Dr. Woodhead also pointed out that, in reference to the question of the structure and importance of giant cells, each authority had his own theory. "It was now becoming evident that each of many of these observers, though describing different conditions, might still claim right on his side. Those who advocate that the giant cell is a lymph space with proliferating endothelial cells around are apparently justified. Then, again, Weigert has proved that a giant cell is nothing more, in some cases, than a collection of cells in which the bacilli are causing proliferation at the margin, fusion and degeneration in the centre—a mass of caseous material in the centre and proliferating cells with bacilli

between them at the periphery resulting. Klein saw the giant cells being formed by the fusion of epithelial cells of the air vesicles. Small blood vessels in transverse section have, like the lymphatics, been described as giving rise to giant cells. Dr. Barrett finds them in the seminiferous tubules in tubercle of the testicle, and Dr. Woodhead has seen them developed in connection with minute bile ducts in the liver, and in the milk ducts and acini in the mammary gland of the cow. In all cases the process Weigert describes occurs, but at different rates and with slightly varying results. The presence of these giant cells affords evidence that *the cells are making a determined resistance against the advance of the bacilli* (see page 58), are giving way slowly, and so limiting the area of caseation. In many cases where the giant cells with their rings of nuclei are best marked, very few bacilli are to be found, as they have been destroyed by the phagocytes at the margin, *i.e.*, the active cells with deeply-stained nuclei. In other cases, however, the bacilli have taken the place of the nuclei at the margin of the giant cell, the boundary line in such cases being determined for a time by the basement membrane of the tube in which the mass is formed."

Dr. Theodore Williams,* quoting Watson Cheyne, Green, and others, classifies the forms of cells which accumulate in the alveoli in lung tuberculisation as—1. Ordinary epithelial cells lining the alveoli. 2. Epithelioid cells. 3. Giant cells. The epithelioid cells "are generally large and spheroidal, about four or five times the size of a leucocyte, containing granular matter and a large, and often oval nucleus, and occasionally a

* "Pulmonary Consumption." By C. J. B. Williams, M.D., etc., and Charles Theodore Williams, M.D., etc. Second Edition, 1887.

nucleolus; more than one nucleus is occasionally seen. These cells are developed from the epithelium, according to Watson Cheyne, and some of them are transformed later into giant cells. . . . In many cases of phthisis, the alveoli are stuffed with the epithelioid cells, some perfect, and others towards the centre undergoing caseation; they are of great interest on account of being the principal haunt of the tubercle-bacilli, which are found in these and in the giant cells, and often in no other lung structure. It is curious to see how the *bacilli are attracted towards them*, and are seen in large numbers both around the cells and within the cell walls. (See p. 58.) The third element is the giant cell, as to the origin of which there has been so much controversy, ending for the most part in the general conclusion that it is epithelial. . . Giant cells are sometimes not found in the very early stage of tubercle development, and appear generally after the products of exudation have become absorbed." "Klein states* that in some of the most rapid cases of acute tuberculosis, no adenoid growth is to be found, but only desquamative or catarrhal pneumonia." He considers that these different appearances of tuberculous matter "represent different stages of the same process, that the fibrinous exudation comes first, and is afterwards absorbed by the surrounding tissue, which becomes infiltrated with fluid and shows distended blood vessels. The exudation is replaced by numerous cells or by giant cells. If the irritation lasts long enough, the small-celled adenoid tissue appears in the alveolar wall."† (See description of tubercle, p. 11.)

* "Anatomy of the Lymphatic System."

† Williams (*op. cit.*).

CHAPTER II.

The Tubercle Bacillus the most dangerous of the several Irritants that set up Tuberculisation—Detailed account of Tuberculisation following Experimental Inoculation of Bacilli—The Eventful Interval between Inoculation and observable Histological Changes—Proportion between amount of Irritant and amount of Cell Proliferation — Sources of fallacy in results of experiments—Clinical demonstration—Symmetrical and unsymmetrical effects of Poisons or Virus on the Mordant-like conditions of tissues—Clinical and Pathological importance of the Mordant-like conditions which determine the domiciliation of Bacilli — Bird's-eye view of the Organism, the Portals by which Bacilli enter it, and the associated intercepting contrivances—Cutaneous Portal, Alimentary Portal, Respiratory Portal, Genital Portal—Tuberculisation due to other Irritants than Bacilli, illustrated by Grit-Phthisis, etc.

GRANTING, then, that the tubercle bacillus introduced into an animal tissue is *one of the irritants* which will cause the formation of the heterogeneous mass which I have described (p. 11), and which, however produced, we agree to call Tubercle; and granting also that it is the most important and dangerous of these irritants; the utmost interest, in a medical point of view, must attach to the earliest steps by which this formation of tubercle —this tuberculisation—advances after the bacillus has entered the tissue.*

The most interesting experiments illustrative of the process of bacillary tuberculisation, are those of Baum-

* See reference to *other irritants*, pp. 29, 30, 41.

garten. They were minutely and accurately made, and observed under circumstances which seem peculiarly free from the chance of error; and, as Mr. Barker says (*op. cit.*), Baumgarten is peculiarly qualified for such work by his profound histological research in other fields, and his experiments have been repeated by many other observers with the same results, and the tissue changes observed have a close parallel in those demonstrated in human organs, as shown by the careful studies by Arnold and others of tuberculisation of the lung. "But as they can be followed in their various stages, and uncomplicated by ordinary inflammatory processes, *only* in experimental inoculations on animals, the latter are taken for illustration. . . . In the experiments in question, a small fragment of caseating tissue is removed from a scrofulous joint or gland, with due precautions against contamination from without, and is inserted in the anterior chamber of a healthy rabbit's eye, through an incision in the cornea (the same operation is performed on a number of other rabbits); . . . one or more of these eyes is excised daily, and placed in preserving fluid for histological examination. Now what is found? (1) *In the first few days absolutely no change is observed*, except the cicatrisation of the corneal wound, and the formation of a capsule of granulation tissue around the foreign body introduced. (2) But from the second day on, a very evident increase of the bacilli is demonstrable *within the inoculated fragment*, until they are present in profusion. (3) From this spot, at which they first multiply, they are now seen to spread rapidly. In the first place they permeate the granulation tissue (capsule) around the caseous particle in enormous numbers. . . . Subsequently they penetrate

into the adjacent parts of the iris and cornea. In the latter (cornea) they spread chiefly along the fresh scar-tissue, a fact of much significance to the operator, explaining as it does the occasional appearance of caseating foci in perfectly healed scars in the skin, perhaps weeks after an operation for deep-seated scrofulous disease. (4) On the fifth day, scattered bacilli are seen in those portions of the cornea nearest to the point of inoculation, *but without, so far, producing the slightest deviation from their normal histological texture.* No traces of other species of organisms are to be seen with or near these immigrants, or throughout the whole iris or cornea. (5) On the sixth day the bacilli are present in great numbers throughout the tissues of the cornea, *which are still, however, practically unchanged.** But at certain points, especially where the bacilli lie in dense groups, certain *newly formed cells* are to be seen having the general appearance of endothelial or epithelioid elements, rather than of white blood or lymph corpuscles (see p. 15). (6) Each day now adds to the hosts of invading parasites, which occupy an ever increasing tract of the tissues of the iris. The nearer to the point of inoculation, the more closely packed are they; the further off, the more scattered. Where the organisms are very sparse no histological changes are to be detected, *but close to the point of inoculation, young tubercles in the form of aggregations of epithelioid cells are now noticed.* (7) A little later, every intermediate form, up to the perfect type of miliary tubercle with their lymphoid and epithelioid cells are to be seen . . . . These formations are constantly found associated with foci of bacillus

* N.B.—It will be seen later on that I call the period from No. 1 to No. 6 "*The eventful interval*" (pp. 44, 58, 59, 81).

proliferation, no organism of any other kind being present. Moreover, in this experiment, no tubercle, not even the smallest, is ever found at a spot free from the presence of the bacilli, or having only a few of them. The amount of cell proliferation always corresponds to the richness in bacilli of these nests of parasites.* (8) Thus developing and extending, the tubercular process advances further, until, on about the tenth or eleventh day, a visible *macroscopic* iritis or keratitis is produced. (9) In *the distant internal organs* of the inoculated animals an entirely analogous process of tuberculisation was observed by Baumgarten. This was best seen in the kidneys, at about the fifth week after inoculation. Here the vessels of the glomeruli were frequently found packed with bacilli, and, *where the latter were spreading into the parenchyma around, typical tubercles were produced.* (10) Large numbers of bacilli were excreted by the kidneys. Similar experiments have been repeated by many other observers, *with cultivated bacilli,* and with exactly similar results. The dosage of microbes was shown by Watson Cheyne materially to influence the rapidity of the development of the disease, a point of the utmost importance as bearing upon clinical experience." Striking examples were given by Mr. Barker, in which similar direct inoculation of tubercle on the surface of the body in the human subject, both in children and in adults, had *accidentally* taken place, and numerous cases have been put on record within the last few years, which place the matter beyond doubt.

In these important experiments, then, it is clearly demonstrated that the tissue changes (*i.e.* tuberculisa-

* That is, it is proportionate to the amount of the irritant, there being no other irritant present but the bacilli.

tion) did not *precede* the presence of the bacillus, but followed it; that the bacillus first became *domiciled in the tissue*, and that then, after a distinct interval (see p. 19)—in the case of the cornea, at least 5 or 6 days—histological changes began, proportionate in amount to the number of bacilli, *i.e.*, to the amount of the irritant.

These appear to be unquestionable facts. But we must remember that in these experiments the bacilli were artificially and intentionally—that is forcibly—inserted into healthy tissues in animals constitutionally healthy. It does not follow that the same sequence of events would occur if the animals were *constitutionally defective*; or if the introduction of the bacillus into the tissue, instead of being by force, were due to some condition of the tissue which made it specially attractive to the bacilli, and hence invited their domiciliation. (See pp. 59, 92.)

It is a clinical and histological fact that there are cases in which the animal organism appears to be patterned out, *as it were with a mordant*, so that when a poison or virus gains admission to the system, it is attracted and domiciled in the patches by an elective affinity—as a mordant fixes the colour of the dyer, and maps out a pattern on the fabric. A strikingly mordant-like condition of the tissues is demonstrated by the simultaneous development of symmetrical patches of disease when the blood is flooded with certain poisons. This indicates that the normal condition of tissues symmetrically distributed is acted upon by a common abnormal influence equally diffused through all. But when, as also happens, the patterning out of the disease is non-symmetrical, it indicates either that the mordant-like condition of the tissue is abnormally localised, or

that the poison or virus is not equally and generally diffused.*

One of the points of greatest interest and importance, clinically and pathologically, is to find out what is this mordant-like condition of tissues which determines the domiciliation of the bacillus tuberculosis.†

In bacillary tuberculisation, both of the above conditions are seen. Most frequently the mordant-like condition is not symmetrical, but indicates some localised tissue fault, or other state, which specially invites the bacillus to domiciliation in the part or parts. Exceptionally, the symmetrical and general distribution of the local disease indicates that the whole torrent of the blood has become infected, the localisation of diseased changes corresponding, not with any abnormal condition of tissue, but with the intensity of normal vascularity, that is to say with the *amount and intensity of the local poisoning.* It is evident that from this cause young and growing tissues, *although normal,* will invite a special incidence of the blood disease.

But in the exceptional cases referred to, although the whole torrent of the blood may be poisoned, and the tissues patterned out with a general symmetrical diffusion of local disease, some want of symmetry is in most instances developed, because it is so rare to find all the tissues of the body equally normal in condition, even in states which may be called "health." Hence, accompanying the general symmetrical diffusion of disease, there are generally found patches of specially intense or extended

* See Mr. Jonathan Hutchinson's remarks, quoting Paget, Budd, and others, in the discussion on Syphilis at the Pathological Society.

† To this I have devoted special consideration further on. See Chap. III.

local disease, giving a want of perfect symmetry in the diseased processes.

It is, therefore, extremely rare to find even acute general tuberculosis without some organ or tissue being unsymmetrically affected by the domiciliation of a special number of bacilli.

Many other complications of a puzzling and confusing character arise in the case of bacillary tuberculosis, in consequence of the number of the portals by which the bacillus may be admitted to the organism, the differences in these portals, and the variety of the contrivances by which its diffusion is opposed and its domiciliation frustrated at the several portals.

It is necessary, therefore, to take a bird's-eye view of the general plan of the organism with regard to these portals and the intercepting contrivances in connection with them. We find a venous system separated inviolably from an arterial system by the lungs, and a great food-supplying apparatus, with the liver placed as peremptorily between the food supply and the pulmonary circulation as are the lungs between the veins and arteries, intercepting every particle of new food that can be absorbed by the portal system of veins—that is to say, the carbohydrates, albuminoid materials, oleinous fat, alcohol, water and any matters soluble in water. Next we find a similarly peremptory arrangement, by which everything absorbable by lacteals and lymphatics is scrupulously kept out of the way of the liver, and conveyed by the thoracic duct to the lungs, but not until it has been submitted to an intercepting system of glands; the materials thus conveyed include all fat not absorbable by the portal system, that is to say, all the solid ingredients of fats emulsified

by the pancreatic secretion; also all worn out tissues, and the products of interstitial nutrition taken up by the lymphatics. Then as regards matters that can be admitted by the respiratory apparatus, we find all sorts of difficulties placed in the way of any foreign irritants contained in the inspired air—to prevent their admission to the alveoli, in which it is intended that only blood and atmospheric air shall come into intimate relationship. Thus there is the long and complicated system of mucous channels, all amply provided with secretory and excretory glands for the expulsion of offending matters before they reach the regions of residual-air; thus, also, the air in inspiratory and expiratory motion is not allowed to traverse these residual-air regions, in which the interchange of respiratory materials is only permitted to take place by the diffusion of gases; thus also, throughout the whole of this important tract, cell proliferation is set up with great promptitude if any abnormal irritant accidentally penetrates beyond the mucous surface. Throughout the cutaneous surface we find, again, elaborate arrangements for the interception and expulsion of abnormal matters before they can reach the blood.

Yet notwithstanding all these fortifications, garrisons and armaments for the protection of the citadel—so subtle is the tubercle bacillus, that it succeeds in gaining admission to the organism by all these portals. Although repelled, expelled, blockaded, imprisoned, killed, cremated, thousands of times, every now and then it succeeds in establishing a domiciliation in the tissues, where it may become encapsuled and indefinitely localised, or, breaking through its prison walls, it may gain admission to the blood, or possibly, under peculiar circum-

stances, it may elude all barriers and directly enter the circulation. Once in the torrent of the circulation, there may begin that mordant-like patterning out of elective or selected tissues which I have already described (pp. 21, 22). But even then it has to run the gauntlet of "the battle of the cells" (see p. 15), by which, according to some, it may at last be destroyed.

Unhappily, it too often happens that it finds out some weak point in the organismal defences, and wins the fatal battle in the end. In the faithful observations of the actual facts of bacillary life, in man and other animals, we find full confirmation of the truth of each item of the foregoing sketch.

Referring to the introduction of the bacillus by the CUTANEOUS PORTAL, Mr. Barker says (*op. cit.*) : "Pfeiffer's case deserves a passing notice on account of its completeness. The patient was a healthy veterinary surgeon, with a good family history, who, while dissecting a tuberculous cow, punctured the joint of his left thumb. [The normal organismal defences of the skin were forced by the puncture.] The wound soon healed, but was followed by induration of the scar, and later by swelling of the whole joint, which underwent the typical changes of a scrofulous synovitis, but without the formation of sinuses. Some months later the patient began to show signs of pulmonary phthisis, which rapidly increased, and he died of this disease a year and a-half after the wound of the thumb. . . . The joint on being laid open, showed all the destructive changes of scrofula both in the bones and synovial membrane; and in the latter, as well as in the broken down material which filled the intervals between the bones, an unusually large number of tubercle bacilli were found. The microscopic

appearance of the diseased tissues was also typically tubercular. Such cases, as those just described, leave no room for doubt that tubercle can be inoculated in any part of the surface of the body, and may spread from the original point of entry throughout the whole system, until a fatal amount of general disease is produced."

With regard to the admission of the bacillus by the ALIMENTARY PORTAL, Dr. Woodhead says (*op. cit.*): "It has, of course, often being objected that if all tuberculous cattle gave tuberculous milk, the human race would run a risk of being rapidly exterminated (see Chap. VI.). But it may now be maintained, as the result of most careful clinical observation, that it is only when the functions of the intestines are interfered with, and when, in consequence, there are temporary or permanent alterations in structure and in the chemical constituents of the fluids and gases in the alimentary canal, that tubercle bacilli can make their way unaltered through the epithelial barrier. . . . . It is not only the intestine itself, however, that is affected by these functional disorders. The mesenteric glands are also placed at a great disadvantage. This may be easily understood when it is remembered that the slightest irritation in any portion is almost immediately followed by changes in the lymphatic channels and glands. Every one is familiar with the peculiar condition of enlargement, congestion, and succulence that is found in such glands. This condition must be looked upon as the result of stimulation; the cells are roused into greater activity, they proliferate more rapidly, and take up the foreign matter: the gland as a whole acting as a kind of sieve. In this process, however, the store

of resistant energy, if such a term might be used, is gradually diminished, and should tubercle bacilli find their way into the gland during or shortly after this extra stimulation, they run less risk of being destroyed, by active epithelioid and lymph cells, than when these cells are not already partially exhausted. This is true in greater degree only in the glands than in the other tissues. Everyone engaged in practice could recall numerous cases in which tubercular disease of bone or synovial membranes, etc., has followed on measles, scarlet fever, small pox, and similar conditions, or in which an attack of typhoid or intestinal mischief of some form has been looked upon as marking the date, soon after which serious tubercular mischief was developed. All this should be considered, leaving out of account the fact that healthy glands might find it difficult to cope with any *large number* of bacilli finding their way through the unhealthy surface. It should be borne in mind, also, that during the growth of the individual these glands are, under normal conditions, called upon to do much more work than during later life, owing to the fact that in the building-up process in the body there is excess of nutritive material required for the tissues, in consequence of which the *secondary digestional function*, which the lymphatic system performs, is provided for; but in imperfectly and badly nourished children this is not the case.* In the first instance the glands act as vital filters, but should the stimulation be too great, there comes a period at which they are destroyed; they degenerate, and may become encapsuled in fibrous tissue, or they may suppurate and so become foci from which other

* See Paper by the Author, "On Fat and Starch in the Nutrition of Children." Reprinted in "Diet and Regimen," Seventh Edition.

areas are affected. Where this sequence is preserved there is a still greater danger at the second stage. So long as the gland is active, the irritant material passed along to it is so modified that it is removed almost inert, but as soon as the degeneration sets in the area connected with that gland is much more liable to ulceration, especially in the case of children." (See p. 97.)

Speaking of the admission of the bacillus by THE RESPIRATORY PORTAL, Mr. Barker says (*op. cit.*): "Ever since the disease (tuberculosis) was first defined, the respiratory passages have been recognised as the point of attack at which the human body most frequently suffers, and clinical and pathological experience has shown that it is from this tract of mucous membrane that the infection most commonly spreads to the other parts of the system. The mode of this infection has so often been demonstrated by experiments on animals as to require only a passing notice here. Not only has it been shown that the injection of phthisical sputa, or crude tuberculous matter, or the bacillus tuberculosis, *derived pure by cultivation* from it, into the air passages will produce typical tuberculosis of the lungs, but it has been observed that even healthy animals of certain species simply living with others thus affected speedily develop the same disease; and again, that healthy animals placed in an atmosphere contaminated by tuberculous dust are soon similarly affected. Not only has all this been proved by many independent observers, but the various stages of the disease, from the first initial lesion of the catarrhal mucous membrane to the fully developed general tuberculosis have been traced step by step in animals thus dealt with. And when we come to consider these experiments, and to compare their results with our clinical experience

of phthisis in the human body and its minuter pathology, we cannot doubt for a moment that its etiology is the same in both cases." (See pp. 94, 95.)

Dr. Woodhead (*op. cit.*) says: "It is now generally accepted that all cases of rapid infective phthisis are the result of the action of the tubercle-bacillus, although it cannot be denied that certain lesions may be produced as the result of the action of *other and non-specific irritants.** In these conditions, however, tubercle is very frequently associated with some other lesions, and it is often extremely difficult to say which changes are due to the one and which to the other. For instance, it has long been held that in *stonemasons' phthisis all the characteristic lesions met with in chronic tubercular phthisis are present*, but my experience of stonemasons' lung has been that, along with the chronic interstitial and arterial changes set up by the stone particles, there are structural alterations which can be accounted for only on the assumption that they are of tubercular [bacillary ?] origin; and in some *few* cases, in confirmation of this, the presence of tubercle bacilli has been demonstrated in certain of the new growths."† In Julius Arnold's observations on the course taken by dust when inhaled into the respiratory tract, he "pointed out:—(1) the very important part played by the walls of the small bronchi and their terminal passages in the disposal of

* See p. 11, and Chap. III.

† Those subjected to the causes of grit-phthisis are, of course, liable to be subjected to those of bacillary phthisis also, and from the condition of their health are specially predisposed to infection; but the concurrence of the two diseases does not prove their identity. See Dr. J. C. Hall, "On the Trades of Sheffield," Second Edition, 1865.

inhaled dust particles. (2) There is the alveolar epithelium, which, under the influence of *any irritant* material, undergoes proliferation more or less rapid—this in certain cases terminating in what we know as catarrhal pneumonia. Here again, in proof of the statement, take what may be seen under the microscope when particles of coloured dust have been inhaled into the lungs. The cells lining the alveoli are seen to be in an active state of division, some are still adherent to the wall of the air vesicle, and *in these* small particles of the pigment may be seen imbedded in the protoplasm. The epithelium in this position is a structure which may be attacked by the tubercle bacilli, just as in it coal or other particles may be found. Passing still further, and following the course taken by the pigment granules, the lymph spaces around the air vesicles are reached, then the lymphatics in the interstices and interlobular tissue, the peribronchial and perivascular lymphatics, and, lastly, the glands at the root of the lung, either directly or by the deep layer of the pleura, over the surface of the lung, and so to the root. As may be seen on reference to a section of coal-miner's lung (exhibited), the pigment—in this instance a material which gives rise to little irritation—is carried to every part of the lymphatic system, and is seen to have accumulated in very considerable quantities along the lines of the septa, around the bronchi and blood-vessels, and in the deep layer of the pleura. On microscopic examination, the pigment *acting as an irritant*, and so giving rise to a slight excess of fibrous tissue in all those various positions, may also be seen. Lastly, the small points of lymphatic tissue which occur at intervals along the lymph channels, first described by Burdon Sanderson, then by Klein, Arnold, and others,

are the seats of pigmentation. *Tubercle formation* (see p. 11), *also may be met with in any of these positions.*" *

With regard to THE GENITAL PORTAL, M. Cornil reported to the Paris Congress, July, 1888, that "he had twice examined a uterus removed during surgical operation; in each case the uterus was tuberculous, in one instance contagion by coitus was out of the question: the disease was seated in the Fallopian tubes; in the other the cervix was attacked, also, the surface of the uterine mucous membrane beneath the epithelium presented tuberculous granulations. As the annexed organs, which were removed, did not present any tuberculous lesions, M. Cornil considered that this was a case of the transmission of tubercle by coitus. In order to ascertain the possibility of this mode of contagion, he introduced some bacilli into the uterine cavity of guinea-pigs [the *Lancet* report says into the vagina]. The first result was catarrh of the cervix uteri, afterwards a considerable increase of lymphatic cells containing bacilli. On the fourth day small tubercles were observed beneath the stratified epithelial cells; the tubercles then invaded the connective tissue between the uterus and the bladder. M. Cornil concludes that sexual intercourse may furnish a means of contagion." (*Brit. Med. Journal*, July 21, 1888.)

* On the subject of grit tuberculosis see (1) Prof. Nothnagel's observations on the passage of cells from the alveoli into the interstitial tissue, related, p. 168, in the second edition of the Author's work On Loss of Weight, &c.; (2) Dr. Osler's (Montreal) description of microscopical examination of miner's lung, "Dobell's Reports on Diseases of Chest," 1876; (3) Dr. Noble Smith's beautiful plates of anthracosis in Dr. Shepherd's Gulstonian Lectures, 1877; (4) Introductory chapter on the perivascular system, in the third edition of the Author's work on Winter Cough; (5) Klein's "Anatomy of the Lymphatic System"; (6) Williams on Consumption, *op. cit.*, 1887, &c.

From the foregoing illustrations we have seen how the bacillus tuberculosis may be introduced by all the chief portals to which I have referred; and this list might, no doubt, be increased by the addition of other less frequent means of access. But those which I have given are sufficient for our present purpose.

CHAPTER III.

The importance of not confining our views of Tuberculosis to Pulmonary Consumption—Opinions of Hughes Bennett, Jonathan Hutchinson, E. Symes Thompson, and the author on the Digestive Organs—Diseases of the bones and joints, illustrative of Bacillary Tuberculosis—Domiciliation, localisation, and dissemination of Bacilli—Nocard's experiments with the muscles and glands of tuberculous animals—Organismal potency and impotency—Excretion of Bacilli—Destruction of Bacilli—Power of Bacilli and their spores to defy all known means of limitation and extermination—Causes of variation in different cases of Bacillary Tuberculosis—Complications due to effects of other irritants—Enumeration of irritants and morbid states associated with the phenomena of Tuberculisation and Phthisis—Initial Loss of Weight and Constitutional Decline discussed—Pre-tuberculous and Pre-bacillary stages—The laws which dominate Habitat—The special food and temperature which determine the habitat of Tubercle Bacilli—Disintegration of proteids by Oxygen and by Bacilli, and the Localities of special incidence—The order of events in Tuberculosis may depend upon the portal by which the irritant enters—General tuberculisation from entry of Bacilli into the vascular system.

It has been one of the most serious obstacles to advance in the study of tuberculosis, that it has been too exclusively identified with tuberculisation of the lungs, *i.e.*, with pulmonary consumption. Thus the area of observation has been narrowed, and the bases for wide generalisations as to cause and effect have been shut out of the investigator's field of vision. I have long striven to divert the attention of the profession from this too narrow view, and as is well known, I have specially

endeavoured to direct the thoughts of observers to the alimentary tract, as one, at least, of the most important sites of *the fons et origo mali.** The late Dr. Hughes Bennett, of Edinburgh, made most valuable contributions in the same direction (1853–59); and Mr. Jonathan Hutchinson, before he was known to fame, published an admirable paper "On the form of dyspepsia which often precedes and attends phthisis" (1855); and in 1864 Dr. E. Symes Thompson published in the "Medical Mirror" a most intelligent article on "Indigestion in early phthisis," beginning with the words, "Deranged digestion is one of the most common deviations from health that ushers in tubercular disease." These and other similar observations and suggestions will be more fully appreciated, now that we have learned how easily the bacillus tuberculosis (unknown at the time those works were published) enters by the alimentary portal, and how easily and fatally, once within these gates, it finds admission to every part of the citadel. (See p. 60.)

On the present occasion, instead of seeking for illustrations of the clinical history of tuberculosis, as is most usual, among cases of pulmonary consumption, I shall take them from the histories of *tuberculous joint disease* as recounted by Mr. Arthur E. J. Barker (*op. cit.*). Mr. Barker's observations on joint disease are applicable to tubercular diseases throughout the body, and the joints afford a very favourable field for their investigation.

Mr. Barker says (*op. cit.*): "It is well known that the disease generally finds its most fertile soil among the

* See Summary of the author's work in Chap. IV., "On Loss of Weight," &c., Second Edition.

very young, probably, because, among other reasons, the vitality of the young cells is unable to cope with the invading organism; and if this be true, we should expect to find that, *with the bacillus once in the blood*, those tissues which are youngest, or in other words approach most nearly to the embryonic type, should be most liable to suffer from its attacks. Now such embryonic tissue is abundant in childhood, and most so at those spots where growth and physiological activity are greatest. . . . If one were asked, then, to suggest the most potent factors in the *predisposition* of joints to tubercular disease, one would be inclined to say, in the first place, the inherent low vitality of the youngest tissues; and, in the second, high physiological activity.* . . . One point, I think, may be accepted as almost certain, namely, that tubercular disease starts more frequently in the bone *alone* in childhood, and in the synovial structures alone in adult life. If this preponderance of primary synovial disease in the adult be a fact, as I think it is, it would be best explained by the *cessation of physiological activity* in the bones, while it *continues in the synovial tissue*, the other factors in the production of the disease remaining unaltered. From the above considerations it would appear that there are several kinds of predisposition to tubercular disease of joints. In the first place a *peculiar debility* may be inherited from phthisical or otherwise unhealthy parents, rendering the tissues of the child generally less capable of repelling the attacks of the parasites (bacilli) than had it come of a robust stock. Next there is the general predisposition of early age, the tissues of the young having less resisting force than those of the mature. Thirdly, certain localities in

* See pp. 21, 22, On Mordant-like Conditions; also p. 47.

the body appear to be particularly open to the attacks of the organism (bacillus), owing probably to the hurried physiological changes involved in rapid growth, which are going on in them—changes which are associated with the presence of abundance of almost embryonic tissue and great vascularity. . . . Finally, there is the predisposition of external injury, using the term in its widest sense. In the case of the joints, injury may render their tissues more open to the attacks of the bacillus tuberculosis circulating in the blood, in several ways acting singly or together. In the first place the violence may cause more or less active inflammation, with exudation of plastic material into the tissues around. Such exudation material resembles embryonic connective tissue, and seems to have very little power to resist the attacks of the organism; in other words, it offers a favourable soil for its growth. This we have seen in the case of the inoculation of the rabbit's eye with tubercle by Baumgarten (see pp. 18, 19, 20), where the bacilli spread more rapidly into the granulation capsule, and along the cicatricial tissue of the corneal wound than in any other direction. Further, the increased blood-flow to an inflamed part would also, *cæteris paribus*, involve a greater supply of bacilli to it than if the vascularity were normal. Again, without any active inflammation, or following upon it, vascular atony may result from violence to a joint, and passive congestion of its tissues be the result, as is so often seen clinically. In this condition not only is the general nutrition of the part damaged, but the slowing of the blood stream in the smaller vessels, amounting often almost to stasis, offers a better opportunity for the deposit of the bacillus in the parts around than is afforded with a normal circu-

lation. And here, too, the greater amount of blood entering the congested area would bring a larger army of organisms to the attack. Finally, injury to a joint will often produce more or less extravasation of blood into its tissues, which remains for a time unabsorbed. With this blood *the bacilli*, too, *escape from the vessels* (see p. 74), and in their new position, withdrawn from the current of the circulation and in a state of rest, they meet with the most favourable conditions for further growth, and soon form large colonies. This last mentioned effect of injury is probably a most potent factor in the determination of the points at which tubercular disease shall start throughout the body, and will best explain its appearance in many unusual situations. Those who are not familiar with Schüller's experiments bearing on this point will do well to give them their careful attention." Parasitic, tubercular or scrofulous disease, "in its modes of spreading by *contiguous infection* through the lymphatics,* and by the entrance of its virus into veins, offers, as we have seen, a close parallel to the modes of generalisation of the so-called malignant neoplasms. . . . . But that, in spite of its capacity for wide dissemination, it may remain for a long time localized in a joint, the rest of the system continuing unaffected, I need not undertake to prove; every clinical observer is thoroughly familiar with the fact.† On the other hand, we must all have been impressed with the experience that in many cases, *after a long*

* See "Grit-Phthisis," pp. 29, 30, 31.

† Prof. McFadyean and Dr. Woodhead found that a cow's udder might be affected with bacillary tuberculosis (as proved microscopically), without the slightest falling off in the general condition of the animal, so long as the disease remained local.—*Lancet*, July 14, 1888.

period of quiescence as a purely local affection, tuberculosis has rapidly become general. We must have noticed that this has usually followed upon some injury to a fully developed cascating focus, or the bursting of some caseous abscess, or, what is perhaps of greater importance for us, as surgeons, to remember, it has followed soon after a surgical operation upon a tuberculised part."

From Wartmann's statistics of 837 resections, it appears that in at least 10 per cent. of the total number of deaths following operation, rapid general miliary tuberculosis supervened. And I may refer here, in passing, to the frequency of a sudden and disastrous descent in *cases of fistula* in tuberculous patients after surgical interference.* Mr. Barker says that "not infrequently in examining a tubercular focus in the cancellous tissue of the end of a bone, we observe that its spread has been prevented, as it were, by a process of sclerosis in the tissue around. . . . If such a sclerosis exist in any given case, we should be careful, in gouging out the diseased matter, not to go beyond this barrier, erected by the natural processes against the spread of the disease," and in a previous passage he says: "Now as long as this limiting layer of plastic exudation remains undisturbed and unbroken . . . we are justified in believing that a very considerable obstacle is offered to the dissemination of the bacilli within it, and that the disease remains, in many cases, almost or altogether localised. But if in any way this barrier is dissolved by suppuration or broken through by violence, a means of escape for the bacilli is provided, and more or less generalisation of the disease takes

* See p. 105, "Dr. Dobell's Report on the Progress of Medicine, 1869."

place. The organisms may be distributed over a wider local area, may be carried along the lymphatics to be *arrested partially or completely in the glands;* or may gain access to an eroded or torn vein, and thus be carried in overwhelming numbers into the general circulation, as shown by Weigert." That bacilli may be got rid of from the general system by excretion, Mr. Barker thinks, has been proved beyond doubt, for they have been found in several of the secretions, and it is also probable that "to a certain extent, when the dosage is small, and the individual vigorous, they can be totally destroyed within the body before they find a suitable soil for their growth."

At the Paris Congress, July, 1888, M. Nocard (see p. 45) detailed some very ingenious experiments, from which it would appear that the muscles have the power to destroy or "digest," as expressed by M. Nocard, the bacilli in such a way that the meat of animals affected with generalised tuberculosis presents but little danger. Thus four cats ate with impunity the flesh of a tuberculous cow, whilst a fifth cat, that had eaten *a lymphatic gland* of the same cow, succumbed in a very short time to experimental tuberculosis. (*Lancet*, Report, August 1888.)

We have now seen that it has been demonstrated in various ways that the tubercle bacillus may be introduced into the animal organism by all the portals that I have enumerated and discussed (Chap. II.), but that by far the most usual and important are the Respiratory and the Alimentary Portals. We have also seen that it may be thus introduced again and again without succeeding in effecting a domiciliation; and that even when it has succeeded in finding a domicile it may

yet be restricted to a limited area for an indefinite time, by the natural protective processes; also, that it may never succeed in penetrating beyond this localised domicile; and that, under certain exceptional combinations of circumstances, the organism is itself capable of exterminating the bacillus after it has effected a domiciliation. But it is, unfortunately, quite certain that so long as any bacilli or spores of bacilli are allowed to continue their domiciliation, there is no security against their breaking through the boundaries of their limitations and extending their operations to an unlimited extent. It appears, also, to be demonstrated that, under certain conditions, the bacilli or their spores may be so introduced from the first as to defy all known possibilities, natural or artificial, of limitation or extermination.

It is the *variations*—

(A.) In the portals of admission;

(B.) In the localities in which domiciliation is effected;

(C.) In the areas of limitation;

(D.) In the means by which escape from these areas is obtained;

(E.) In the potency or impotency of the organismal efforts to imprison or expel the enemy;

(F.) In the auxiliary forces raised for the assistance of the regular forces;

(G.) In the consequences of success or failure in these operations;—*that account for the variations* in the clinical and pathological features of different cases of tuberculosis, due alike to the introduction of the bacillus tuberculosis.

But these clinical and pathological variations are still further multiplied and complicated by the fact that

other bodies besides the tubercle bacillus may enter the animal organism by the same portals, and set up processes closely resembling, if not analytically the same as, those due to the tubercle bacillus itself (see Chap. II.). Thus, as I have stated—quoting from what I wrote in 1880, just before the discovery of the bacillus—any irritant capable of setting up in the living tissues "a foreign mass made up of hyperplasia of adenoid tissue, epithelial and connective tissue proliferation, angioplasia, and other attempts at repair of disintegrated tissue, and the results of degradation and decomposition of these new formations" may produce symptoms which, when fully established, are clinically and pathologically so analogous that the differentiation of the original irritative cause can hardly be made clear without the aid of chemistry and the microscope, and of a critical enquiry *into the etiology of each case.* I attempted to describe* the various irritative causes of the heterogeneous mass called tubercle when affecting the lungs, as "a large and important group embracing all foreign substances which find their way into the peri-vascular and peri-alveolar tissue of the lungs, and by their irritation there" set up the changes just detailed; and I enumerated the following items as "some of the principal constituents of this important group." (See p. 76.)

"(*a.*) Lampblack, coal, steel, stone, flint, and other substances inhaled by workers in various trades. (See Chap. II.)

(*b.*) The products of inflammatory destruction of tissues.

(*c.*) The products of catarrhal affections (especially in scrofulosis).

(*d.*) The *débris* of tissues disintegrated by the extra-

* "On Loss of Weight," Second Edition, pp. 168-170.

vasation of blood, and possibly the *débris* of the blood so extravasated.

(*e.*) Albuminoid materials disintegrated by oxidation in true tuberculosis (hypothetical)," (see Chap. IV., on special sustenance of bacilli). And I added that this hypothetical item (*e.*) " is the only one in the list which involves that *initial loss of weight* characteristic of the true first stage of consumption. In my opinion this is the irritant which, in true (or pure) tuberculosis, sets up the hyperplasia of adenoid tissue, the cell proliferation, angioplasia, etc., and their results, so well described by Portal, Virchow, Sanderson, Rindfleisch, Charcot, Malassez, and others. But whereas they,—ignoring the clinical fact of *initial loss of weight*,—place these processes *first* among the pathological changes of tuberculosis, I, on the other hand, believing it to be essential to take cognizance of the initial loss of weight and of strength, give precedence to the (hypothetical) disintegration of albuminoid matter by oxidation *in situ*, of which the hyperplastic, proliferative, angioplastic and other changes are but the effects." In 1885, in a chapter on "Disease Tracts" *—written after the announcement of Koch and Baumgarten's discovery—in the following enumeration of the "morbid states connected at different stages with the phenomena of phthisis," I added the bacillus to the list, as follows :—

"(1.) Abnormal physiological states, principally connected with the digestive and assimilative processes, producing constitutional decline. (The true first stage.)

(2.) Disintegration of tissues and organs dependent upon abnormal nutritive processes. (See Chap. IV. special sustenance of bacilli.)

* " The Medical Aspects of Bournemouth," p. 205.

(3.) Abnormal growths of adenoid, epithelial and connective tissues, and the results of abortive formative processes. (Tubercle. See p. 11.)

(4.) Catarrh, hyperæmia, inflammation, gangrene.

(5.) Decomposition of the *debris* of disintegrated tissue, of the products of disease, and of the abortive attempts at repair above referred to.

(6.) General septic disease, or septicæmia.

(7.) Secondary local diseases of septic origin (auto-infection).

(8.) The presence of microbes (bacilli.)"

It will hardly be disputed that any one of the foregoing list of interstitial irritants is capable of setting up the formation of an abnormal conglomeration of matter such as we are accustomed to call "tubercle," and that, unless it should be proved that "giant cells" have some typical connection with the bacillus tuberculosis, (see pp. 11—16), we have no means at present of determining chemically or microscopically whether or not the irritant, in any given case, was the bacillus, except by discovering the presence of the microbe itself or its spores. (See pp. 12—29.) Nor will it be disputed that the list I have given fairly represents "the morbid states (including the bacillus) connected, at different stages, with the phenomena of phthisis."

But now that the existence of a bacillus tuberculosis is undoubtedly established, it may well be questioned whether the item (*e.*) hypothetically described as "albuminoid materials disintegrated by oxygen"—which, as I have said, is the only one in the list which involves the initial loss of weight characteristic of the True First Stage of Consumption,—ought not to be henceforth considered as inseparable from the bacillus itself, the first

palpable effect of which is molecular disintegration (see Chap. II. and Chap. IV.).

But we are met by this difficulty, that, notwithstanding all the elaborate work spent upon the bacillus, no clear evidence has yet been produced that its presence in the animal organism excites any symptoms of *constitutional decline*, until, *like other irritants*, it has set up local disease.* We have seen (Chap. II.), that it has been experimentally proved that, in *bacillary tuberculisation*, no tuberculous matter is formed until a *definite interval* has elapsed after the local domiciliation of the microbe.

But we have not learnt by this experimentation that any symptoms are excited by the bacillus during this *Eventful Interval* (see p. 19). In Baumgarten's experiments, on the fifth day after inoculation scattered bacilli were seen in those portions of the cornea nearest to the point of inoculation, but *without* so far producing the slightest deviation from the normal histological texture. On the sixth day the bacilli were present in great numbers throughout the tissues of the cornea which were still, however, practically *unchanged*. But at certain points, especially where the bacilli lay in dense groups, certain newly formed cells were to be seen having the general appearance of epithelial or epithelioid elements. A little later every intermediate form, up to the perfect type of miliary tubercle with their lymphoid and epithelioid cells were to be seen (see pp. 18-21).

On this subject we have also the important observations of Klein, (*op. cit.*) that "in the most favourable cases (anthrax and tuberculosis) a single organism introduced

* See this subject more fully discussed in Chaps. IV. and V.

into a *suitable locality* in the animal body will be capable of starting readily a new brood. But in other cases it is necessary that an appreciable number of the organisms be introduced in order to start a brood. . . . The period between the time of introduction of the organism into the body (blood, skin, mucous membrane, subcutaneous tissue, lungs, alimentary canal), and the production of the new brood large enough to produce a definite effect, locally or generally, corresponds to the incubation period of the disease, and, as is well known, there is in this respect a great difference in the different diseases. . . . In tuberculosis, after the introduction of the bacillus tuberculosis into the subcutaneous tissue, the nearest lymph glands* show the first signs of swelling and inflammation after one week, or even later, and the general disease of the internal viscera does not follow until one, two, or more weeks have elapsed. This is also borne out by the observations of the behaviour of these bacilli in artificial cultures."

The result of all these observations and experiments is to bring us back to the great clinical question (see p. 2): In what relation does the tubercle bacillus stand to the initial loss of weight and of strength—that pre-tubercular stage so fraught with interest in its connection with preventive treatment—the constitutional decline of bacillary consumption? (See pp. 42, 43.)

In considering this question we must ask, (A) on the one hand:—

(1.) Are these general symptoms due to the presence of the bacillus in the general blood current?

* See Nocard's "Experiments on Cats," p. 39, and "Alimentary Portal," p. 26.

(2.) Are they antecedent to its localisation?

(3.) Are they subsequent to its localisation, and due to its local ravages before these have advanced to a stage at which we can detect them by our present diagnostic appliances?*

And, on the other hand, (B) we must ask:—

(4.) Are these general symptoms altogether antecedent to the domiciliation of the bacillus either in the blood or in the tissues?

(5.) Do they proceed from a local defect or from a constitutional defect?

(6.) Do they indicate a constitutional or local defect which *invites* or *attracts* the bacillus to effect a domiciliation—which, in fact, provides those mordant-like conditions referred to in pp. 21, 22, those feeding grounds for the bacilli of which I shall soon have to speak?†

(7.) Or do they indicate a constitutional or local defect which—without specially "inviting or attracting" the bacillus—simply renders the general or local organismal defences impotent to repel the invader? (See pp. 15, 16, 26, 34, 83.)

These are burning questions in relation to practical medicine; and it is evident that the proper preventive and curative treatment of "The True First Stage of Bacillary Consumption," must be utterly dependent upon their being properly answered.‡

Crookshank says (Second Edition of Manual) that "bacteria are never to be found in the blood in health. The organs removed from a perfectly healthy animal,

* See Chap. V.

† See "Habitat," p. 48, and "Sustenance," Chap. IV.

‡ See also List of Questions, pp. 3, 4, and Answers, Chap. V.

with the necessary precautions, into sterilised media, can be kept indefinitely without undergoing putrefaction or giving any development of bacteria." (See also Watson Cheyne.)

Klein says (*op. cit.*): "One of the most important points, and the most difficult of comprehension, is the power of the pathogenic organisms to *resist the influence of the healthy tissues of the living animals*, a power which . . . . is not possessed by the *non*-pathogenic organisms. A careful analysis shows at the outset that this power of the pathogenic organisms is not possessed by them indiscriminately, for while a particular species is in some animals capable of overcoming the influence of the living tissue, *i.e.*, to multiply and produce the particular disease; in other animals it is not capable of doing this, and hence the animal remains unaffected—it is said to be *not* susceptible to the disease." "Now," says Klein, "where are we to look for this difference in behaviour? . . . . The tissues *per se* cannot be said to possess any inimical action on the organisms. The living condition *per se* can also not have this power, since we see that the power to overcome the influence of the living tissue is precisely the *great distinguishing character of pathogenic organisms.*" (See p. 35.)

I fear we must conclude from the results of the forcible experimental introduction of bacilli into the bodies of healthy animals (see p. 21), that the organismal forces alone, even in health, are not, as a rule, capable of either expelling or destroying the bacilli, or of preventing their domiciliation, when introduced under certain conditions; and that to effect these ends, artificial help is needed of every kind that science and art can devise. And this gives the most emphatic importance to the identification

and treatment of the *pre-tubercular stage*, and still more to the *pre-bacillary stage*.

We must not allow the microscopical finesse of the subject of microbes and the difficulties in observing their habits to cause us to forget that,—throughout the vegetable and animal world, observations, made under circumstances less perplexing than those connected with molecular and atomic rearrangements of matter, have always taught that THE FIRST LAW WHICH DOMINATES HABITAT IS THE PRESENCE OF APPROPRIATE FOOD, AND THAT THE SECOND LAW IS THE PRESENCE OF APPROPRIATE TEMPERATURE.

"Where the carcass is, there will the eagles be gathered together," is but the crude enunciation of the first vast law of habitat throughout the world. The converse is alike true, for where the carcass is *not*, the eagles will *not* be gathered together.

What, then, is the food and what the temperature which together determine the habitat of the bacillus tuberculosis? (See Chap. V.)

We have now many reliable data from which to answer this question. Speaking of the artificial cultivation of bacteria out of the animal body, Klein says (*op. cit.*): "The materials which I now prefer for the cultivation of pathogenic and septic bacteria are: (1) Broth (beef) and peptone; (2) Solid nutritive gelatine; (3) Solid Agar Agar (Japanese isinglass) peptone meat extract; (4) Fluid and solid serum of blood; all faintly alkaline." "In 'fractional cultivations' (the attempt to isolate by successive cultivations the different organisms that have been growing previously in the same culture) . . . most probably only one species (of

organism) in each tube, *i.e.*, the one that grows best in this particular medium and at this particular temperature, will be found to have increased to an enormous extent, while the others have made little or no progress." Speaking of the tubercle bacillus especially, he says: "The bacilli grow well at a temperature varying between 37° C. (98°·6 Ft.) and 39° C. (102°·2 Ft.) . . . Owing to the fact that tubercle bacilli require for their growth high temperatures, it is evident that, unlike some other pathogenic organisms, they do not thrive in the outside world in temperate climates." It is therefore "necessary, in order to study more accurately the life history of the septic as well as the pathogenic organisms" out of the body, to provide an artificial *Incubator* by which the requisite temperature may be maintained.

Now it most unfortunately happens that there is no "incubator" so perfect as the animal body itself; for in it a temperature of 98° to 99° Ft. is constantly maintained, and this temperature is readily raised to 102° or 103° Ft. by the disturbances of disease; while it is equally unfortunate, as it is equally true, that all the selected artificial "cultivation materials" above enumerated, are analogues of the far finer media provided in the blood, the tissues and the secretions of living animals.

Another most important consideration in relation to habitat is the fact that "all bacteria, pathogenic and non-pathogenic, require for their growth and multiplication oxygen, which they either obtain from the media in which they grow, and which oxygen is dissolved in those media, or if this is consumed or absent it is obtained by the bacteria in the process of the chemical decomposition of the carbohydrates and proteids present." (Klein, *op. cit.*)

When treating of the disintegration of proteids by oxygen ("On Tuberculosis," 1866, and "On Loss of Weight," 1880), I showed that the relations of oxygen to the circulating media of the animal body are most similar in the lungs and in the lymphatic system—precisely those regions which are most liable to the incidence of bacillary infection. I pointed out that "there is no part of the body traversed by fluids essentially the same as those which traverse the lacteals, mesenteric glands, thoracic duct, and pulmonary artery, and there are no other parts of the body in which the relations between pancreatised fats, albuminoid tissues and oxygen are so similar as in the small intestines and mesenterie system and in the lungs. . . . In the lungs the oxygen is in the air of the alveoli, and the material to be oxidized is in the capillaries of the alveoli; in the small intestines and mesenteric system the oxygen is in the capillaries of the glands, and the material to be oxidised traverses the glands. Hence we find that the disintegration of proteids by oxygen (or by bacilli?) which occurs first in the lungs, in both children and adults, is next most frequent in the small intestines and mesenterie system—except when it falls upon the bronchial glands of children, an exception suseeptible of explanation consistently with the foregoing statements."

Now, if we regard the bacillus as the disintegrating factor we shall find that it may occupy the same relative positions with regard to the disintegration of proteids as the oxygen in the above description, *i.e.*, if admitted by the respiratory portal (see Chap. II.), it will be in the air of the alveoli; if admitted by the alimentary portal, it will be in the lacteal and lymphatie vessels; if it enters the general blood current (see Chap. V.), it will

be in capillaries of the lungs and glands. In each case, therefore, the bacillus would have two separate directions from which to operate upon the proteids; but the symptoms would necessarily differ in the following manner:

If introduced by the respiratory portal, *local lung disease* would *precede* constitutional decline (see List of Questions, Chap. I. and the Answers, Chap. V.). If introduced by the alimentary portal into the lacteal and lymphatic system, *local gland disease* would be coincident with disturbed nutritive functions, and constitutional decline would be *coetaneous* with it, or might *precede* it by a short interval in consequence of obstruction of the absorbents by the bacilli before tuberculisation was set up (see Chap. II.). If introduced to the general current of the blood primarily (see Chap. V.), any or all of the organs of the body might be attacked simultaneously or in the order of their mordant-like elective affinity for the bacilli (see Chap. II.). It seems reasonable to suppose that this general tuberculisation might be preceded by a period of initial constitutional decline, due to the presence of the bacilli in the blood. But, as I have more fully pointed out (Chap. V.), we have at present no definite evidence that such is the case, or indeed that any abnormal symptoms are produced by the bacillus till it has established tissue domiciliation (pp. 19-21).

In addition to the above, there is yet another position to be considered with regard to the bacillus in the blood, viz.: that, after it has effected a local domiciliation and has remained for a longer or shorter time as a purely local affection, it may make its way into the vascular system and overwhelm the patient with general tuberculisation, as pointed out by Weigert, Wartmann,

Barker and others (see pp. 37 and 39). But as a rule, this will only occur after the local disease has sufficiently advanced to have set up a *secondary* constitutional disease; so that it does not come under the head of *initial* loss of weight and strength characteristic of the True First Stage of Consumption (see List of Questions, Chap. I., and Answers, Chap. V.).

CHAPTER IV.

Bacillary Tuberculosis considered as—1. A Primary Blood Disease. 2. A Primary Local Disease. 3. A Secondary Blood Disease. 4. A Secondary Local Disease. 5. A combination of the above with Suppurative and Septic Processes—Effects of Vitalised and Non-vitalised Irritants compared—Special virulence of Bacilli due to locomotion, multiplication, and demand for sustenance—Difficulty of assuming the mental attitude essential to a just consideration of the Life History of Microbes—Suggestions relating to the "Eventful Interval" after Inoculation—Tubercle piled up by measures of Organismal Defence—Battle between Bacilli and Cells—Laws of Habitat and Domiciliation of Bacilli—Sustenance of Bacilli, its nature and sources—The Feeding Grounds which determine the Habitat of Bacilli hypothetically accounted for—Constitutional Decline premonitory of Bacillary Tuberculisation, in relation to the Feeding Grounds—Discoveries and views of A. M. Brown, Gautier, MacMunn, Peter, and Selmi—Myohæmatins, Histohæmatins, Enterochlorophyl, Ptomaïnes, Leucomaïnes, Alkaloids, Extractives—The physiological state ceases to be normal the instant continuity of vitalised transition gives place to molecular death.

From all that has gone before we have learnt that bacillary tuberculosis may, in all probability, be:—

1. A primary blood disease, bacilli entering the blood first. (See this discussed, Chap. V.)

2. A primary local disease, bacilli introduced into the tissues first.

3. A secondary blood disease, bacilli first domiciled in a part and then getting from it into the blood.

4. A secondary local disease, bacilli first entering the blood and then becoming domiciled in a tissue.

5. A combination of all the above conditions, with the addition of suppurative and septic processes.

In its position as a primary local disease (No. 2), and so long as it remains localised, it takes its place beside the other local irritants enumerated at pp. 41-42, capable of setting up the formation of the heterogeneous foreign mass which we call "tubercle." (See p. 11.) But the bacillus can never be divested of a specific character which other irritants do not possess, viz., *its vitality.* This, at all times and under all conditions, adds to its dangers as a local irritant the far greater danger of its becoming *generalised.* In addition to this its vitality gives it an extraordinary virulence, over all other irritants, from its inherent capability of multiplying and gathering force as it advances. Doubtless, it is this quality which makes it set up cell-proliferation, etc., etc., so much more promptly, and in so much greater abundance than any of the lifeless irritants.

But in addition to these causes of virulence there is yet another item to be considered which is more important than all, viz., that in common with every other vitalised entity it *must have sustenance.*

Whether the bacillary army is bivouacking or advancing, it must have sustenance; and as it does not carry its rations with it, they must be obtained on the road. As the bacillary host is continually reinforcing itself by internal multiplication the demand for this sustenance is ever multiplying at a compound rate.

The questions—(1) What is the nature of this sustenance? (2) By what modes is it obtained?—bring us to the very core of the subject of this treatise.

We have become so familiar with these minute

vitalised entities (microbes) by the perfection of our chemical, microscopical and spectroscopical appliances, that it is almost as difficult for the mind to formulate just ideas of their relation to the world in which both they and we "live and move and have our being," as it is to realise and weigh the conceptions that "the ponderable universe consists of detached stellar groups like that of our milky way, and, like that of the Nebula in Andromeda; . . . . that the vast celestial spaces between these groups are beyond the confines of our universe—that they are, in fact, outside the universe of ponderable matter;"* "that electro-magnetic actions are due to a medium pervading all known space; that it is the same medium as the one by which light is propagated; that non-conductors can, and probably do, . . . transmit electro-magnetic energy, and that, by means of variable currents, energy is propagated into space with the velocity of light." (Hertz's Experiments.)†

The subject of Mr. Crookes's address to the Chemical Society (1888), is intimately related to that of the sustenance of microbes; "The possible existence of bodies, which though neither compounds nor mixtures, are not *elements* in the strictest sense of the word, but '*meta-elements*,'—that what we are accustomed to consider an element is an aggregate of slightly dissimilar atoms of the same primordial material, an elementary group." ‡

The contemplation at once of the vast conceptions of the "detached stellar groups" of the astronomer, the "electro-magnetic ether waves" of the physicist, and of

* G. S. Storey, Chemical Society, 1888.

† Prof. G. F. Fitzgerald, Bath Meeting of British Association, 1888.

‡ Report in *Chemist and Druggist*, April 7, 1888.

the *infinitesimal* "elementary groups" of the chemist, brings before the mind the extremes of subtlety which modern refinements in chemistry, spectroscopy, and electricity have imposed upon our investigations of force and matter.

It is evident that in considering so subtle a question as the sustenance of microscopic vitalised entities, the first thing we have to do is to divest our minds of all our customary ideas of nutrient materials, as they occur in their coarse and ponderable forms. Sir Henry Roscoe* says, "Before chemists can answer as to the course of nature's action, they must find out the *molecular* constitution of the various bodies by synthesis in the laboratory"; and Professor Thiselton Dyer replies that "the building up of substances from simple elements (in the laboratory) does not throw any light upon their occurrence in the living body." Professor Gladstone, speaking of colloids and crystalloids points out that "although proteids are colloids, the substances of which they are made up are crystalloids—which are believed to be very *complex* molecules." Professor Kühne† considers that "*movement* is an attribute of protoplasm both in animals and plants, and it is impossible to have a cell which does not preserve it at some time or other. This movement is a spontaneous activity which may go on in opposition to gravity, and which overcomes frictional resistance. Its cause can only be internal, and can only consist in chemical processes taking place within the protoplasm itself;" while Professor Gardner* "has considerable difficulty in thinking of protoplasm as a substance, and is of opinion that the phenomena of life

* British Association Meeting, 1888.

† Croonian Lecture, Royal Society, 1888.

consist in an infinite arrangement and rearrangement of an exceedingly complex system of molecules, and that protoplasm will ultimately be found to be the development of energy, and nothing tangible"!

Now what I wish to suggest,—as an almost inevitable conclusion from all that we at present know of the life history of the Bacillus, and the conditions of its existence,—is that, in seeking to find out the nature of its sustenance and the means by which this is obtained, it is to the subtle regions of elements, meta-elements, protoplasm, and complex arrangements and disarrangements of molecules, that we have to direct our search, not among such coarse materials as even the minutest cells that the microscope can detect or the ordinary formulated atomic compounds of the chemical laboratory.

A startling evidence of the necessity for this conclusion, in the present state of our knowledge, is found in the statement of Klein (*op. cit.*), that "what the chemical influence of pathogenic organisms on animal tissues may be *is not yet known;* and even when they grow outside the body, *i.e.*, in artificial cultures, it is not yet known what their chemical effect on the nourishing material is, except that, as is the case with all other organisms, putrefactive and pathogenic, they continue to grow and multiply as long as there are present the necessary substances, *i.e.*, until the medium is exhausted."* I have already (Chap. II.) related the fact that when tubercle bacilli are inoculated into the normal tissues of a healthy animal, no detectable histological changes

* "Pathogenic organisms cannot thrive if proteids or allied compounds and certain organic salts are absent."—Klein, *op. cit* See "Habitat," p. 48.

occur for several days. But that these days are *secretly eventful** in the highest degree, is shown by the rapidity and activity of the generation of microbes and of the histological changes which immediately follow. It is impossible to avoid the suggestion that these *eventful* days are occupied in the disarrangement of molecular matter—perhaps in the disassociation of the constituents of Crookes's "meta-elements or elementary groups" (see p. 55); perhaps in the disintegration of the complex crystalloid molecular constituents of proteids (see p. 55)—in order to provide the necessary sustenance for the multiplying army of invading microbes before it commences its advance. It is only after it has been so provided with the necessary sustenance, and so enabled to reinforce its numbers, that its attack is *recognised by the invaded tissues* (see p. 19). And, then, in the attempts to bar the progress of the advancing foe, all those measures of organismal defence are set in motion (see pp. 15, 16), which pile up the heterogeneous mass which we recognise under the name of tubercle. (See p. 11.)

When we find the bacilli swarming into the newly-formed cells (see p. 16), it is highly probable that they are attracted there in the *pursuit of the molecular débris which constitute their means of sustenance*, and which have been taken up by the cells in their attempts at the reconstitution of normal elements and at the construction of defences against the microbes. "It is curious to see," as Dr. T. Williams observes (p. 16), "how the bacilli are attracted towards them, and are seen in large numbers both around the cells and within the cell walls."†

* The *Eventful Interval*, see p. 19.

† See "Discussion on Giant Cells," Chap. I.

According to Koch the bacilli, by-and-bye, disappear again from the giant cells—probably when there is no more to be got out of them. Klein says (*op. cit.*), that "where in a tissue pathogenic bacteria find the suitable conditions for growth and multiplication, they can do successful battle against the amæboid cells, but where these conditions do not obtain, the bacteria linger and die, and, like other particles, can be swallowed up by the amæboid cells. That the presence of bacteria in the protoplasm of amæboid cells *does not indicate* that the former are being removed or destroyed and their action neutralised by the latter, but exactly the contrary, is proved in Koch's septicæmia of mice, in bovine tuberculosis, in leprosy, and in other diseases."

Now, with reference to that short interval which I have called "*eventful*,"—which follows the experimental inoculation of bacilli and precedes observable histological changes, and of which I have suggested that it is employed in the important process of preparing sustenance for the bacilli in their new domicile—we must remember, as I pointed out (p. 21), that in these experiments the bacilli are artificially and intentionally —that is, *forcibly*—inserted into healthy tissue in animals constitutionally healthy. It does not at all necessarily follow that this interval would occur, if the introduction of the bacilli was *spontaneous*. On the contrary, if we call to mind the first law that dominates habitat (see p. 48), viz., the presence of appropriate food, we shall conclude that the spontaneous domiciliation of the bacillus, *i.e.*, its selecting any special locality for its habitat, must be due to its finding there its appropriate means of sustenance.

If, then, we are to regard as essential to the appro-

priate sustenance of the bacillus, a condition of molecular disintegration of crystalloids and proteids, and the like; we are confronted by the important question:—How are we to account for the existence of such a condition in the organism, independent of the presence of the bacillus; that is to say, antecedent to its domiciliation? Are there any known circumstances favouring or inducing such a condition? (See Chap. V.)

It is a singular coincidence, if no more, that the result of my long course of investigation into "the nature and cause of tuberculosis,"* (before the discovery of the bacillus), was to lead me to suggest as an hypothetical explanation of the loss of weight and of strength—the slow constitutional decline—antecedent to the formation of tubercle, and characteristic of the worst form of consumption—that a disintegration of proteids by oxidation *in situ* took place in consequence of abnormal conditions in the digestive and assimilative functions. (See p. 34.) And I showed that the points of special incidence of tuberculisation, in these cases, were precisely those in which such disintegration of proteids would necessarily occur; and that the order of frequency in which these special sites would be affected, corresponded with the order of frequency in the incidence of tuberculisation, as shown by clinical and pathological observations. (See pp. 50, 51.)

Supposing, then, for argument sake, that my hypothesis were correct, it would account for:—(1.) A period of constitutional decline, antecedent to bacillary tuberculisation; (2.) The establishment of feeding-grounds, provided with the proper sustenance for bacilli; (3.) The consequent selection of these special sites by the

* *British Medical Journal*, January and February, 1866.

bacilli for their habitat; (4.) The mordant-like action of tissue faults in relation to bacillary domiciliation. (See pp. 17, 21, 33, 42; also Chap. V.)

I may venture to recapitulate here the words in which I stated my hypothesis regarding the disintegration of proteids, etc., by oxidation *in situ* :—

"The blood becomes deficiently and defectively supplied with fat elements from the food; is unable to afford those required for direct combustion; does not replace those taken up during interstitial nutrition; but, on the contrary, takes up more, to compensate the deficient supply from the food. This having gone on up to a certain point, the fat elements of the albuminoid tissues are seized upon, and these tissues are minutely disintegrated in the process. . . . The word tissues must be understood to include *albuminoid materials employed in the construction and repair of tissues*, for it is probable that the elements may be waylaid during the process of tissue formation (oxidised *in situ*) . . . These *débris* of the combustion of tissues set up adenoid hyperplasia, angio-plastic, epithelial, and other cell proliferation, and all that follows in their track. In this relationship they fall into the ranks of other causes of similar changes. . . . My hypothesis, based as it originally was upon clinical data, framed to explain these data, and entirely consistent as it is with these data, must take precedence of all hypotheses in which they are disregarded . . . No hypothesis but this attempts to explain how *loss of flesh precedes all other changes.* I am astonished that so palpable a clinical fact as the loss of flesh, premonitory of tuberculisation, should have been entirely lost sight of by all modern pathologists. . . . All their ideas seem

based upon the basic clinical fallacy that tubercle precedes constitutional decline instead of following it."

"According to my view, therefore, the order of events is as follows: (1.) Deficiency of fat in the blood (this deficient supply of fat from the food is necessarily felt *first* in the blood of the pulmonary artery, presented for aëration in the lungs); (2.) Oxidation and disintegration of albuminoid matters (proteids) *in situ*, with the consequent production of molecular *débris*. (See pp. 63-9.) (3.) Hyperplasia of adenoid (lymphatic) tissue, the effect of the *irritation caused by the molecular débris* of the disintegrated albuminoid matter." (Pp. 166-70, "Loss of Weight," 2nd Edition.)

Now, it must be observed that this last item (3) is the position in which the discovery of the bacillus may find a place. (See pp. 5, 42.) Supposing, as I have said, for the sake of argument, we assume items 1 and 2 to be correct, and supposing (see pp. 58, 63-9) that the special sustenance of the tubercle bacillus is the molecular *débris* of proteids and their constituent crystalloids—then we find the necessary food provided for the bacillus (see p. 48, the first law dominating habitat); and hence, instead of saying, in item 3, that the molecular *débris* sets up adenoid hyperplasia, etc., etc., and causes tubercle, we should have to say that it *determines the habitat of the bacillus*, and thus causes tubercle, the bacillus, and not the molecular *débris*, being the irritant.

I do not assert that this is correct; but it is only fair to point out in this place that it presents still, as I thought it did in 1880, a good solution of the problem:—What causes the constitutional decline antecedent to "the worst form of tuberculisation," or, as we must now say, to "Bacillary Consumption"?

But, instead of attributing this molecular *débris*—this hypothetical sustenance of the bacilli—to the defective absorption of fats, and consequent combustion of proteids *in situ*, as suggested in the foregoing hypothesis; in the light of our present knowledge, we may, possibly, find an explanation of the existence of some such materials, and a consequent "abnormal physiological state"—constituting the special form of constitutional decline antecedent to bacillary tuberculisation,—in the spectroscopic discoveries of Dr. MacMunn, of Wolverhampton, referred to in my treatise on asthma (1886), which he has since further extended;* and in the discoveries of Selmi, Gautier, Peter, and others, elaborated in the exhaustive treatise of Dr. A. M. Brown,† and ably and lucidly epitomised and commented upon by Sir William Aitkin in his recent brochure "On the Animal Alkaloids, the Ptomaïnes, Leucomaïnes, and Extractives, in their Pathological Relations" (1887). Sir William Aitkin says:—

"Gautier has shown that in the dead animal tissues,

* "Researches on Myohæmatin and Histohæmatin." By C. A. MacMunn, M.A., M.D. Proceedings of Royal Society, No. 240. Read Oct. 19, 1885.

Ditto, ditto, Transactions of Royal Society, 1885. Read Nov., 1885.

"Observations on the Chromatology of Actiniæ." Transactions of Royal Society. Read January, 1885.

"Further Observations on Enterochlorophyl and allied Pigments." Transactions of Royal Society. Read April 30, 1885.

"Further Observations on some of the Applications of the Spectroscope in Biology, etc." Proceedings of Birmingham Philosophical Society. Read April 8, 1886.

† "Contributions to the Study of the Cadaveric Alkaloids, the Ptomaïnes and Leucomaïnes; their Physiological and Pathological significance in relation to Scientific Medicine." By Dr. A. M. Brown. 1886.

processes of putrefactive decomposition set in by which certain alkaloids are elaborated from the proteid substances, which by the late Selmi, of Bologna, have been called 'ptomaïnes.' But Gautier has further shown that in the living animal tissues, and that by virtue of their *vitality*, certain other alkaloids are elaborated which are analogous to the 'ptomaïnes,' and these he has named 'leucomaïnes.' Still further . . . he has demonstrated that in the living animal economy there are elaborated certian azotised uncrystallizable substances, which are as yet undetermined, which we call 'extractives,' or 'extractive matters,' and which are quite as unknown as the *x*, *y*, *z*'s of an algebraical formula (Dr. H. L. Veale). The nature of these 'extractives' has, therefore, still to be found out; but this much we know of them: that while we are assured that the 'ptomaïnes' are toxic, and that the 'leucomaïnes' are also toxic, these unknown 'extractives' are more toxic or poisonous to the system than either. . . . It was at first supposed that these animal alkaloids differed in their nature from the organic alkaloids formed by vegetables, and various reactions had been given to distinguish them. More recent researches, however, especially those of Brieger, appear to show that this distinction can be maintained no longer; but that the animal and vegetable alkaloids are similar in their chemical constitution, and that they are both products of albuminous or proteid decomposition; and that some, at least, of the so-called 'ptomaïnes' are identical with vegetable alkaloids.

"'We may now indeed regard alkaloids,' writes Dr. Lauder Brunton* . . . . 'as products of albuminous decomposition, whether their albuminous precursor be

* "Pharmacology and Therapeutics."

contained in the cells of plants and altered during the process of growth, or whether the albuminous substances undergo decomposition *outside* or *inside* the animal body, or by processes of digestion as by unorganised ferments.' ('Pharmacology and Therapeutics,' 3rd Edition.) . . . . But Gautier has further shown by his researches, extending from 1881 to 1886, that the animal alkaloids are also a necessary product of *vital physiological processes* he having obtained from the secretions of living beings alkaloid bodies, having poisonous properties—results which have been confirmed by M. Peter.

"They have shown that about four-fifths of our disassimilations are the result of transformations within the body, comparable with the oxidation of alcohol; and that the remaining one-fifth of the disassimilations are formed at the expense of the living tissues themselves, 'free of all demands on foreign oxygen.' In other words, a fifth part of our tissues live after the manner of ferments; that is, they are anaërobious or putrefactive as to their life. Hence the possibility of alkaloids being thus formed within the living organism *independent of bacterial fermentation* is quite within our conception." (See p. 60.)

Speaking of the clinical, pathological, and practical aspects, Dr. Aitkin continues: "According to the different sources of poisoning or intoxication (as it is technically called), there are correspondingly different indications, signs, or symptoms capable of classification as below: (1) Poisoning by the 'extractives' is attended by hy*per*thermia. (2) Poisoning by the 'animal alkaloids' is accompanied by 'hy*po*thermia.' (3) A combination or succession of hyperthermic and hypothermic phenomena may become manifest, according to the combination or alternation of poisoning by the deleterious physio-

logical products or their antagonistic action (Dr. A. M. Brown). . . . .

"There is in these researches still further disclosed the fact that in this *auto-infection*, this spontaneous or self infection of the living organism by the 'alkaloids' and 'extractives' of its own formation, there is *no question of quality*, but simply one of *quantity* to be considered, by reason of the essential physiological source and action of the poison. In other words, the healthy living organism may become poisoned (more or less slowly) by the accumulation within itself of deleterious substances normally elaborated, but imperfectly or defectively eliminated. Hence the slow and insidious onset of much ill-health, and from which recovery is correspondingly slow." (*Op. cit.*)

It is not probable that in "the normal physiological state" which we call "health" (see my lectures on "The Germs and Vestiges of Disease") any particle of the living body, even the most subtle molecule, element or meta-element, can properly be said to be *dead* until the instant at which it is to be thrown off by an excretory organ. A rough analogy of this may be drawn from the "fall of the leaf," which in a healthy tree is due to a vital act; the vitality of the leaf is not extinct until it is cast off.

Perfectly healthy histogenesis consists in the insensible, uninterrupted passage of molecules from one vitalised condition into another, up to the point of excretion as a non-vitalised excrement. The instant this continuity of vitalised transition gives place to *molecular death*, the physiological state ceases to be normal, and the first step of disease is taken. All sorts of contrivances

are provided in the organism—and for the most part successfully—for stopping disease at this first step—THIS INITIAL STAGE.

It is conceivable that the *raison d'être* of microbes is the removal of these initial steps of death, and that these devitalised molecules constitute one at least of those means of sustenance—those feeding grounds which determine their habitat. (See pp. 48 and 54-58.)*

* These remarks and quotations regarding "Ptomaïnes" and "Leucomaïnes" were first written after reading Sir W. Aitkin's brochure in December, 1887. I am gratified to find, on reading Dr. Latham's Hunterian Oration (*Lancet*, Oct. 20, 1888), that he has arrived at conclusions in many respects so similar to mine, that had I read his before I wrote mine, or had mine been published before he wrote his, we might easily have been supposed to have copied each other. Thus Dr. Latham says :—"I wish to draw the inference that, in the living animal organism, owing to slight departures from the normal nutrition of parts, arising probably through nervous agency—the trophic nerves—various substances, such as the extractives or alkaloids, will be produced, which, if not eliminated or neutralised, will lead to pathological changes in the system, *absolutely and entirely independent* of any *bacterial action*. [See p. 65, Dr. A. M. Brown, quoted by Aitkin.] "Or it may be that these poisonous substances act specially as irritants of epithelial structure, causing an overgrowth of epithelial cells, just as we know will result when any other irritant is applied to these tissues, and so the soil is prepared for the lodgment of the bacillus." . . . (See my remarks on other irritants, pp. 19, 29, 41, and 62.) "It may be suggested that when the bacilli have gained a footing in . . . effete or enfeebled tissue, while absorbing from it the constituents of their own protoplasm, they may give rise to or secrete poisonous products or ferments capable of still further destroying or weakening the surrounding tissue or cellular elements, and so obtain fresh food on which they can thrive." [See my remarks on the Eventful Interval, pp. 45, 46.] "Perhaps by further investigations into the chemical changes produced by bacilli, the constitution of the proteid molecule, complex as its nature is now regarded, may be unravelled and its properties better understood ; we may be able to discover, possibly, what poison it is

which the bacillus secretes, or how it is formed, or from what constituent of the proteid molecule it is derived. The other constituents of the molecule may perhaps furnish the antidote, or a poison antagonistic to that produced by the bacillus ; for we know that poisons are antagonistic to each other—atropine and muscarine for example."

Crookshank says ("Manual," Second Edition) :—"Though we may accept as a fact the existence of pathogenic organisms, we are not yet in a position to assert the means by which they produce their deleterious or fatal effects. Many theories have been propounded."

Klein (*op. cit.*) concludes that:—"The most feasible assumption, and the one borne out by observation, is that owing to the multiplication of the (pathogenic) organisms, certain chemical changes are produced in the blood and tissues, or that a special ferment is created, which sets up the anatomical changes characteristic of the particular disease."

Dr. R. W. Philip, of Edinburgh, believes that he has separated and identified a toxic product from phthisical sputa.

Dr. Philip's conclusions (*British Medical Journal*, Jan. 28, 1888) are as follows :—"1. In view of the work of Koch, it is impossible to avoid admitting that a causal relationship exists between the tubercle bacillus and the phthisical process. 2. The mere predication of this relationship is not sufficient in explanation of the clinical facts and the generally fatal termination of such cases. 3. The usually received explanations of the *modus moriendi* in phthisis are insufficient. 4. It appears probable that the lethal influence of the bacillus is due to the production thereby of certain poisonous products. 5. Clinical and experimental evidence appears to indicate *that the morbid secretions from the respiratory surfaces afford a good medium for the growth of the tubercle bacillus, and, presumably, for the elaboration of such products.* 6. Such a product is separable from the carefully selected and prepared sputum. 7. This product is possessed of well-marked physiological properties, being eminently toxic to frogs, mice, and other animals. 8. The toxic properties of the product are, speaking generally, depressant. 9. More particularly they include a marked depressant influence on the heart. 10. This depressant influence seems to be exerted through the medium of the cardio-inhibitory mechanism. 11. The toxic action of the product is more or less completely opposed by atropine. 12. The amount of the product which may be separated appears to bear a distinct relation to the abundance of the bacillar

elements present. 13. Absorption of the *poisonous product most probably occurs by way of the lymphatic circulation.*"

It will be observed that with the exception of the "substances, such as the extractives or alkaloids" (of Gautier and Peter), which "lead to pathological changes in the system absolutely and entirely independent of any bacillary action" (Latham), all these poisoning theories presuppose the domiciliation of the bacillus in the animal; and, therefore, although they may have value as accounting, as Dr. Philip suggests, for the *modus moriendi*, they in no way account for a *prebacillary constitutional decline*, or for the *predisposing conditions which determine the habitat* of the bacillus, which are the points in which we are now most especially interested, an which I am attempting to explain.

CHAPTER V.

Consideration of the question whether the presence of Bacilli in the organism can produce constitutional decline antecedent to local signs of their domiciliation—Is there a portal by which Bacilli can spontaneously gain direct admission to the blood?—Experiments of Cornil on the alimentary mucous tract—Acute Miliary Tuberculosis due to auto-infection from Bacillary centres, by ulceration of pulmonary veins, by thoracic duct, by venous radicles of glands—References to Barker, Coats, Ponfick, Wartmann, Weigert, Woodhead—Infection by Filaria Sanguinis referred to—C. J. B. Williams and J. Hughes Bennett on Cod Liver Oil and Pancreatic Emulsion—Does the portal by which Bacilli enter the organism determine the division of Pulmonary Consumption into two great classes, one which must be Bacillary, and one which may be due either to Bacilli or to other irritants?—Answers to questions suggested in Chapter I.—Four ways of accounting for constitutional decline antecedent to Pulmonary Tuberculisation—The hypothesis of "Lowered Vitality" a refuge for the destitute—The "Eventful Interval" may possibly be prolonged, but will not account for Predisposition—We must renounce belief in Predisposition to domiciliation of Bacilli, or accept the existence of some Prebacillary state determining the habitat of the parasites—Possible incompetence of present means of diagnosis—The first steps towards death precede abnormal physical chest signs and the presence of Bacilli or *débris* of air-cells in sputa—Repetition of warnings given by the author in 1858 and 1867—Importance of treating the "True First Stage," and difficulties of diagnostic differentiation.

If we reject the foregoing hypothetical explanations of the initial loss of weight and of strength—the constitutional decline—characteristic of The True First Stage of Bacillary Consumption, we appear to have only the following alternative, (1.) To reject as fallacious the well-

known clinical fact that constitutional decline may precede bacillary tuberculisation; (2.) To assume that it is due to the presence of the bacilli in the organism before they have produced any local signs of their domiciliation. (See p. 19.)

Let us then carefully consider what probability there is of this assumption being true.

It has been amply demonstrated (see *ante*), both clinically and experimentally—

1. That if tubercle bacilli gain admission to a part, and are there imprisoned—all access to other parts and to the torrent of the circulation being barred—no constitutional decline will be produced while they so remain. (See pp. 37, 38.)

2. That such localised domiciliation of bacilli, having set up tuberculisation in the part and subsequent destructive changes, may, if the affected part is one important to health, lead to a sort of constitutional decline of a *secondary* nature.

3. That if the bacilli escape from their local imprisonment and become disseminated in a number of localities, the tuberculisation and consequent destructive changes, thus extended, will set up constitutional decline of a *secondary* nature even "unto death."

4. That if the bacilli, under either of the above conditions, get access to the torrent of the circulation, they overwhelm the vital power, and cause rapid death by acute miliary tuberculosis. "Weichselbaum ascertained that in acute miliary tuberculosis of man the blood contains the bacilli." (Klein, *op. cit.*) (See pp. 46, 51.)

But in all these cases the constitutional decline is a *secondary* state, not an *initial* one antecedent to the establishment of local disease.

In order to account for initial constitutional decline antecedent to the local domiciliation of the bacillus, we must revert to the idea of the existence of the bacilli in the general circulation as a *primary* condition.

It is true that such a condition has been assumed and stated by some observers. And in the enumeration (p. 53) of the different modes by which the animal organisms may be attacked by bacilli, I have mentioned "a primary blood disease—the bacilli having entered the blood first."

But when we come to inquire more closely into the evidences that such a state is possible—except as the result of the forcible introduction of bacilli in experiments (see p. 21)—we shall find that they are almost *nil*. In the first place we have to find out a portal by which the bacilli can enter the blood direct. The existence of this has not yet been proved; but such discoveries as those of Lewis in 1872, and of Bancroft in 1876, with regard to the "Filaria sanguinis hominis," and the marvellous manner in which *even so large a parasite* may find its way into the blood, should make us hesitate to deny the possibility of the bacillus finding some easy means of access to the general circulation. At the Paris Congress, July, 1888, it was suggested that the bacillus can migrate through mucous membranes without hindrance by the organismal defences.

"M. Cornil communicated some interesting experiments on contagion of tuberculosis through mucous membranes. The cultures of the tubercle bacillus, introduced into the intestine, penetrate very quickly into the mucous parietes. In introducing into the œsophagus of guinea-pigs a few drops of tuberculous cultures, he always observed, at the end of four days, submucous tuberculous

lesions, with rapid generalisation without lesion of the epithelium." (*Lancet*, August 4, 1888.)

It has, also, been suggested that the bacillus, having been introduced with food into the alimentary tract, may find its way by the lacteals into the mesenteric glands, and, through some defect in the intercepting functions of these glands, may enter the thoracic duct and thus get into the general circulation. (See Chap. II.)

These are suggestions which still wait for further evidence before they can be considered to be established as facts. But even supposing that, by either of these suggested portals, the bacilli can gain primary admission to the blood current antecedent to localisation; all that we as yet know, clinically or experimentally, of the effects of bacilli in the torrent of the circulation is against the idea that they can exist there without rapidly setting up symptoms of dangerous illness. The most reliable examples at our disposal, from which to judge of the effects of bacilli in the general circulation, are cases in which they have entered it by auto-infection, from the breaking-up of local bacillary centres, either by natural processes or by the intervention of the surgeon, as pointed out by Mr. Barker and Dr. Woodhead, and in the statistics of 837 re-sections of tubercular joints collected by Wartmann. (See p. 38.)

It is true that "the bacilli, though found in the blood in such cases, do not become *active* until they come to some part of the circulation in which they can make their way into the surrounding tissue." (Woodhead, *op. cit.*) But it is also true that this opportunity very soon occurs, and that they lose no time in taking advantage of it to permeate all the tissues and organs. Thus Dr. Woodhead says (*op. cit.*): "Acute miliary tuberculosis

must be looked upon as the result of spreading of the infective material directly by the blood-channels. The demonstration of this fact was first accomplished by Weigert, who, in a series of several cases of acute miliary tuberculosis, was able to determine the presence of ulceration of the pulmonary vein. The process being similar to that in or near the wall of a bronchus. . . . Ponfick had first supposed that the bacilli might pass from a tuberculous thoracic duct into the venous trunks, and thus into the general circulation. It is probable that both observers were correct, and that both forms may occur. Coats further points out that a limited distribution of tubercle by the blood may be due to the passage of bacilli into the minute venous radicles in the glands in which tuberculous changes are occurring. That bacilli are found in the blood has been now frequently demonstrated (see p. 71), and quite recently several cases have been recorded in which general tuberculosis has come on after hæmorrhage in patients suffering from apical phthisis. This is a matter of greater interest when it is borne in mind that all these cases of acute tuberculosis were developed in from seventeen to twenty-five days—just the period given by Koch as that required for the development of tuberculosis when produced experimentally."

It does not seem very probable, then, that a slowly encroaching constitutional decline, unaccompanied by signs or symptoms of localised disease, can be due to the presence of bacilli in the general circulation.

I think it is more probable that bacilli, entering by the alimentary portal, and thence gaining admission to the lacteal and lymphatic systems, may so impede normal nutrition by these important channels as to lead to

constitutional decline, which in this case would be *antecedent* to localised pulmonary tuberculisation. The effect of such a condition would, in fact, be similar, as regards loss of weight and of strength, to the effects of arrested or perverted pancreatic digestion, as detailed at pp. 60-62. In the case of the Filaria sanguinis, referred to p. 72, such obstruction of lymphatic glands and vessels by the ova of the parasites does produce constitutional illness. But we know how obscure are the physical signs of abdominal tuberculisation, even when accompanied by wasting to the extent of marasmus in children.* And it is conceivable that constitutional decline, due to this cause, might precede for a considerable time the domiciliation of the bacilli in the lungs and the development of the physical signs of *pulmonary* tuberculisation.

Such a mode of attack would be accompanied by the symptoms of defective assimilation of fats; and would be relieved by cod-liver oil—absorbable by the portal veins, and hence independent of the mesenteric system—and by fats emulsified by pancreatisation—so as to facilitate their absorption by the defective and choked lacteal system, or by those portions of it still capable of exercising their functions.

Up to the present time, no therapeutic remedies have been able to compete with pancreatic emulsion of fat and cod-liver oil in the power to restore loss of flesh, and to prolong life in abdominal and pulmonary tuberculosis. Dr. C. J. B. Williams says, "I have no hesitation in stating my conviction that this agent (cod-liver oil) has done more for the consumptive than all other

* See the Author's paper "On Fat and Starch in the Nutrition of Children," in the Seventh Edition of "Diet and Regimen."

means put together" (*op. cit.*, p. 336), and the late Dr. J. Hughes Bennett said, "What then is really required is not oil added directly to the blood, but oil digested and emulsionised by the pancreatic and other intestinal fluids; a truth which has induced Dr. Dobell to recommend that before administration it should be mixed with pancreatic juice." ("Reynolds' System of Medicine," 1871.)

But it is of the utmost importance to bear in mind that, while the hypothetical explanation which I have just suggested is consistent with known clinical histological and therapeutical data, it *limits bacillary consumption with initial constitutional decline* to cases in which the bacilli have gained admission to the organism by the alimentary portal. (See Chap. II.)

It may be, then, that the mode of attack—the portal by which it is made—in this way rules the division of pulmonary consumption into two great classes; both of which may be due to bacillary infection, but one of which—the one characterised by initial loss of weight and of strength (constitutional decline)—can only be bacillary, whereas the other may be due either to bacilli or to one of a long list of other irritants. (See p. 41.)

The result of my investigations with reference to such a division into two great classes, published in 1880, before the discovery of the bacillus, holds good in the light of subsequent research, as we shall see by the following list of answers, which I then published, to the complicated set of questions which I had propounded, and which I have detailed in this treatise at page 3.

In quoting these answers I have now interpolated references to the Bacillus at appropriate places. "It is found (see my tables), That :—1, in a large number of cases of pulmonary consumption, local disease is preceded by

constitutional disease *by an unequivocal interval.*—Constituting Class 1.

"2. In a certain number of cases, local disease precedes constitutional disease by an unequivocal interval.—Constituting Class 2.

"3. These two classes must be considered as absolutely distinct throughout.

"4. There is no stage at which these two classes meet on common ground. Class 1 is always *plus*, as regards Class 2. (That is to say, the constitutional decline, which in Class 1 precedes the local disease, is always an extra factor to the factors in Class 2.)

"5. There is a stage in which the constitutional disease sets up the local disease in Class 1." This stage I described in 1880 as consisting in the setting in of the hypothetical oxidation of albuminoid matter. And supposing our more recent views to be correct, we must now describe it as consisting in the dissemination of the bacilli from the lacteal and lymphatic systems, and the tuberculisation caused by their presence in the lungs. (See pp. 60-62.)

"6. The first stage of the local disease in Class 1 is not the same as the first stage of the local disease in Class 2." This answer will now only hold good for cases in which the local disease is not due to bacilli. Where the pulmonary disease is due to the introduction of bacilli by the respiratory portal, the first stage of local disease will be substantially the same in both classes.

"7. Starting from this point, the subsequent *constitutional symptoms of decline* are *not* alike due to the effects of the progress of the local disease. In Class 1 they are due to the progress of the constitutional disease *plus* the effects of the local disease, and in Class 2 they are due *only* to

the effects of the local disease. The first will always therefore, *unless arrested*, beat the second in the race of constitutional decline."

"8. The local disease in Class 2 does not *per se* set up a state of constitutional disease of the same nature . . . as Class 1."

"9. A new constitutional disease of special character may be set up by the local disease in Class 2, and the local disease of Class 1 is competent to set up a constitutional disease of the same special character as in Class 2. In both these cases the special constitutional disease is due to the absorption of the products of the local disease." We may now add that when both cases are bacillary, there will not only be 'absorption of products,' but dissemination of bacilli.

"Finally, as already stated, the symptoms of constitutional decline which *follow* the establishment of local disease in each set of cases (Class 1 and Class 2) are not 'devoid of special character, and only such as accompany the progress of any local disease proceeding to a fatal issue,' but are of *a distinct and special character*, due, in Class 1, to the defective supply of fat to the blood, etc., etc." Or, as now hypothetically suggested, to the choking of the mesenteric system with bacilli. "While in Class 2 the symptoms of constitutional decline are due to irritative hyperplasia and proliferation. . . . . exhausting discharges and absorption into the blood of morbid products." And we must now add, that when the local disease is in both classes due to bacilli, the special secondary constitutional disease may also be due to the dissemination of the organisms.

"It is competent for the original cause of constitutional decline in Class 1, viz., arrested passage of fats into the blood"—either by defective pancreatisation or by bacilli,

to be overcome in the course of the disease, the bacilli and their effects being cleared from the mesenteric system, or the defective pancreatisation cured; and thus the cause of loss of weight antecedent to local disease in the lungs, characteristic of Class 1, may be removed; in which case "Class 1 will approximate in *its later stages* to Class 2." *

It will be seen from the foregoing recapitulation that pulmonary phthisis preceded by constitutional decline is to be accounted for by either of the following ways:—

1. By the direct admission of bacilli to the general circulation, and some *unknown influence* there, antecedent to their localisation in the lungs. Of which we have no reliable evidence. (See pp. 72, 73.)

2. By a defective absorption of fats, due to choking, etc., of the lacteal and lymphatic system by bacilli, admitted by the alimentary portal, and afterwards conveyed to the lungs—there to become domiciliated and produce tubercle. (See p. 72.)

3. To a defective supply of fats, due to defective pancreatisation (see pp. 60-62), leading to the molecular disintegration of proteids, the *débris* of which may constitute the special food for tubercle bacilli, determining their habitat in the parts where such *débris* are to be found—such parts, as we have seen (p. 57), being especially and primarily the lungs and mesenteric glands.

Or, 4. To the production of such *débris* in the manner described by Selmi, Gautier, MacMunn, A. M. Brown, Aitkin, and others (as already set forth, pp. 63-69). "The possibility," says Brown, "of their being formed within the living tissues *independent of bacterial* fermentation is quite within our conception."

* "Loss of Weight," Second Edition, p. 206.

Now it will be observed that all the foregoing hypotheses attempt to explain, not only *some form* of constitutional decline antecedent to tuberculisation, but a "*special form.*" If we dispense with the conditions of speciality, and simply try to explain the occurrence of *some or any* form of constitutional decline, we may, of course, fall back upon the old, well-worn, often refurbished suggestion, of "lowered vitality." I have long regarded this as "the refuge of the destitute hypothesis." But it may be that, even now—after all that modern discovery has disclosed—we are still so destitute of better explanations, that some may think it best, to adopt the manœuvre of covering our retreat in the smoke of "lowered vitality"; which may be made to account for anything, anywhere, that tends towards death.

But what we want is an explanation of a *special form of tendency towards death*—an explanation of why the tubercle bacillus effects a domiciliation in this person and not in that, an explanation of what determines its incidence on this organ rather than on that. (See pp. 21, 35, 46, 47, 48, 49.)

If we cannot, at present, find an explanation which is entirely satisfactory, we had much better stand bravely by our guns and confess that we are waiting for fresh ammunition, than keep up a fire of blank cartridges under the delusive name of "lowered vitality." I am afraid we can detect a tendency to the firing of these blank cartridges—to a falling back upon "the refuge of the destitute hypotheses"—lurking among some, even of the most learned and scientific, recent disquisitions on tuberculosis—evidently unperceived by their accomplished authors.

It may be humiliating to our *amour propre* as medical

men — but nevertheless it must be faced — to suggest a source of fallacy, which involves our own incompetency, as possibly at the bottom of all our difficulties in accounting for "a special form of constitutional decline" antecedent to tuberculisation. It must be regarded as still within the limits of possibility that constitutional decline special to bacillary tuberculosis is, after all, not antecedent to the first local inroads of the bacillus, but only antecedent to the *evidences* that it is present and at work *discoverable* by our present powers!

What if that "eventful interval" to which I have more than once referred (see pp. 19, 44, 58, 59), which, in our experimental inoculation of tubercle bacilli, elapses between the insertion of the bacillus and the first evidences of its work detectable by the most minute chemical and microscopical tests — that interval which, I have suggested (p. 58), may be occupied in the molecular disintegration of proteids, crystalloids, etc., by the microbes, to provide sustenance for the gathering forces of their invading army (p. 59)—what if that interval should, under certain circumstances, be capable of *prolongation* until a definite observable "special form of constitutional decline" is developed, no local indications of disease being detectable by our present means of examination during the life of the patient!

It must be remembered that we seldom have opportunities of *post mortem* examination of patients in this *pre-tubercular* state, and that when, by some accident, such an opportunity occurs, it is only men with such special qualifications and appliances as are possessed by MacMunn, Watson Cheyne, Crookshank, Klein, and a few others, in this country, who would be competent to

eonduct sueh a subtle investigation as would be neeessary for obtaining reliable results (see p. 63).

But even supposing the interval between the introduetion of the baeillus and the eommencement of tuberculisation to be eapable, under certain eonditions, of suffieient prolongation to aceount for an observable constitutional decline, this will not provide us with the essential eonditions for determining habitat (p. 48); it will not account for the predisposition of one person and not of another, or of the same person at one time and not at another, to the domieiliation of the baeillus. On the contrary, the supposition neeessarily involves the *forcible* introduction of the baeillus, as in the cases of experimental inoculations, not a spontaneous eleetion or seleetion of a certain habitat. Therefore we must either throw up our belief in the existence of *predispositions to bacillary domieiliation*, or aeeept some hypothesis of a *pre-bacillary* state determining the habitat of the parasite, whether or not sueh a state is demonstrable by any appreeiable diagnostie symptoms.

Although I have suggested the possibility that the speeial form of eonstitutional deeline anteeedent to our *detection* of any local domieiliation of bacilli may, nevertheless, be due to the baeilli, and that the secret of the non-deteetion of their work may be found in the ineompetenee of our present means of diagnosis; it will be seen, by all that has gone before, that this *is not the opinion to which I myself incline.*

I still think it most probable that the "speeial form of eonstitutional decline" is due to *pre-baeillary conditions*—conditions by whieh the neeessary sustenanee of the baeilli is prepared, and henee their habitat in the animal organism determined, in aceordance with the laws whieh

determine habitat throughout the vegetable and animal kingdoms. (See Chaps. III. and IV.)

This brings us back, at last, to the practical object with which I commenced this disquisition (see p. 1), viz., To attempt some sort of articulate answer to the call of the medical practitioner: "Tell me, in the state of our knowledge *this day*, what is the best thing I can do to repel this enemy at our gates? What ought I to prescribe for this patient suffering from that 'special form of constitutional decline' constituting the true first stage of bacillary consumption?" (See p. 46.)

It is said that an old stage coachman, expatiating on the advantages of the road over the rail, exclaimed, "If you have an accident on the road, why there you are! but if you have one on the rail, why, where are you?"

If we adopt this graphic form of comparison in the case of consumption, we cannot find even the coachman's comfort. For I fear we must say, "If you find the bacillus, why where are you? for you do not know how to expel it; and if you don't find the bacillus, why, still, where are you? for it may be there!"

Speaking of the detection of bacilli in phthisical sputum, Dr. Troup says (*op. cit.*), "Naturally it is only where the breaking up of the original reaction products has begun and opened a way outwards for the bacillus that it will be found. . . . In acute miliary tuberculosis it will *not* be seen in the beginning, and it is here and in all suspicious cases that a thorough quest for *elastic tissue* should be instituted." But elastic tissue cannot be present till the disintegration of *formed tissue* has taken place, and this is *far too late* for the diagnosis of "the true first stage," the stage which precedes any such tissue changes. Besides, as Dr. Troup is obliged to admit, "one cannot

say" that the elastic tissue "is only present in the spit of phthisis—other destructive changes cause its appearance."

In the discussion on Dr. Percy Kidd's paper, at the Medical and Chirurgical Society,* Dr. Heron said, "I wish to emphasise with Dr. Kidd the importance of *examining cases again and again.* Very often the bacilli will remain undetected for weeks, and after examining the sputa during four, five, or six weeks, they will be found in large numbers."

To which Dr. Douglas Powell very aptly replied:—"While the discovery of bacilli in the sputum is of positive value as a qualitative test, showing the existence of tubercle, the *absence* of bacilli is not by any means of equal negative value, unless, as Dr. Kidd has said, the case be watched for many weeks; and in the course of these many weeks most of the *physical signs* will have developed."

Why! in the experimental inoculation of bacilli into healthy animals which I have recounted, we have seen that "four or five weeks" is time sufficient to allow the bacilli to become domiciled in every organ of the body! (See pp. 19, 45, 74.)

Klein says (*op. cit.*):—"After the introduction of the tubercle bacillus into the subcutaneous tissue, the nearest lymph glands show the first signs of swelling and inflammation, *after one week*, or even later, and the general disease of the internal viscera does not follow until *one, two,* or more weeks have elapsed. This is also borne out by the behaviour of these bacilli in artificial cultures."

It is evident that the detection of elastic tissue and

* "On the Value of the Tubercle Bacillus in Clinical Diagnosis." By Percy Kidd, M.D., and H. H. Taylor, F.R.C.S. (May 22, 1888.)

of bacilli in the sputum is, like the detection of physical chest signs, all too late. We must be far in advance of all this if we wish to anticipate the first steps towards death (see p. 66)—if we wish to prevent or abolish the molecular feeding grounds, before they have led to the domiciliation of the bacillus (pp. 46-8).

Thirty years ago—at a time when the physical signs of auscultation and percussion were expected to detect everything connected with diseases in the chest—I published the following warning. And if we now add to our list of "physical signs" the presence of elastic tissue and bacilli in the sputum, the warning is almost as much needed to-day as it was then. Concluding a chapter on the "Diagnosis of Early Phthisis," I wrote :—

"This absence of reliable signs of the earliest stage of tubercular deposit cannot be too forcibly impressed upon the young practitioner, who, with creditable zeal, is too apt to think, and naturally prone to hope, that by sufficient diligence, experience, and care, he may insure that no tubercle shall escape his searching examination. In this belief he will be often led to fancy that he has detected the presence of tubercle where it does not exist, and to assume its absence while it really lies concealed.

"This is the great disappointment which every man has to encounter who studies and practises physical diagnosis. That upon which he had set his heart—to detect the first shadow of consumption, scarcely yet resting upon a life, before it is too late to drive it back—is the very dream which experience will most surely dissipate.

"If physical diagnosis could detect consumption as soon as the first few spots of tuberculous matter were deposited in the lung, with the same certainty that it

detects pneumonia or a cavity, we might well be content to sacrifice for this all that it could do besides. But that it cannot, in its present state, accomplish this, and that there is no good reason to suppose that it ever will accomplish it, need surprise no one who will think calmly on the subject. It is not probable that the physical properties or the functions of a portion of lung-tissue should be sufficiently affected by a few scattered gelatiniform granules, to produce any recognisable signs of their existence. There may not be a dozen tubercles deposited in the whole lung; nay more, there may not be one single microscopic speck deposited, and yet the disease may be working its *stealthy inroads on vitality*. We shall not, then, place implicit confidence in the physical diagnosis of early phthisis, but—while not neglecting this, while seeking from it all the aid it can give—we should exert our most acute observation of symptoms and diligently search into the *histories of cases*, in the hope that we may thus encounter some herald of the coming foe."*

When writing upon this subject again, in 1867, I insisted, with all the force I could put into words, that what has heretofore been called the "Premonitory" stage of consumption ought to be at once recognised as "The True First Stage."

"It is utterly absurd, when applied to practical medicine, to make a disease begin at a point at which serious structural change takes place, but which it is impossible to identify with certainty during life. The truth is that these divisions in the course of the disease have been made by the morbid anatomist in the *post mortem*

* "Demonstrations of Diseases in the Chest, and their Physical Diagnosis," 1858.

room, not by practical pathologists and physicians in the chambers of the sick. It is not surprising, therefore, that they are worse than useless in practice . . . It is evident that the use of the terms 'premonitory stage' and 'first stage,' as now employed, can have no sense except on the assumption that the *formation of tubercle constitutes the disease;* and thus a false pathology is directly impressed on the mind of both physician and patient."*

Speaking of the difficulties which the pathologist has to encounter in determining the histological origin and course of tuberculisation, Dr. Woodhead says (*op. cit.*), "It would seem at first sight to be an easy matter to determine at once in what tissue the tubercle has originated in any special case. In the lung, however, where the tissues are so delicate and so complicated, and where, in consequence, the changes are so rapid, *this is not the case;* and it is only in exceptionally favourable cases that the mode of origin and spread can be at all satisfactorily demonstrated. Further, the variety in the life histories of individual tubercular growths at one time rendered it a matter of considerable difficulty to arrive at any definite understanding of tubercular processes, especially of those associated with pulmonary phthisis. The anatomical structure in the various forms being so absolutely defined in the earlier stages of the growth, it was difficult to bring into a common group forms which differed so widely from one another, not only in naked eye, but in microscopic appearances."

But if difficulties of diagnostic differentiation are so great when the clinical histories of the cases have been played out, and when the affected tissues are subjected to

* "On the True First Stage of Consumption," 1867.

examination in the laboratory; how incomparably greater are those difficulties when the cases are in their "*initial stage,*" before the microscopist or the chemist has any materials for examination, and when local and general clinical phenomena have scarcely dawned out of the regions of imagination!

This is a period so eminently critical to the patient, that the turning of the balance for life or death may depend upon the placing of a few grains of evidence in the right or the wrong side of the judicial scales. Yet this is the period when the physician ought definitely to begin his treatment of the true first stage of bacillary consumption.

CHAPTER VI.

The object of correct pathology and diagnosis is not diagnosis, but treatment—In such subtle abnormal physiological states as the True First Stage of Consumption, the first rule of treatment is "to keep on the safe side," the side of precaution—The scope of treatment must be wide enough to embrace the possibility that the patient's state is *ante*-bacillary or *post*-bacillary—Every function must be called to its account with a jealous regard to the strength of the patient—Much knowledge, skill, and tact are required—There is no rough and ready rule—Every case must be taken on its own merits—Common sense should rule—Pause and think before you act—Duties of medical men—Difficulties in exercising educated common sense, because of the obstacles erected by patients and their friends—Dr. Clifford Allbutt's advice—Importance of vigilant medical supervision—"Let well alone" is a wretched axiom for a physician; he should ever be on the watch "to do better"—Intercurrent treatment must be subservient to that founded on the basic nature of the disease—Details of treatment discussed—Climate, Diet, Regimen, Medicaments.

THERE can be no doubt that we are right in having come to the conclusion in recent years that a medical man should be a scientific man. But we shall have lost more than we have gained if, in becoming scientific, he ceases to be practical. The fascinations of scientific research must never lead us to forget that the object of correct pathology and diagnosis is not diagnosis, but treatment. Our object in diagnosing The True First Stage of Bacillary Consumption in its earliest dawn, is not merely to signalise a scientific triumph, but to enable us to quash it in its dawn by proper treatment, and thus

to discharge the highest functions of the medical man—to prevent human suffering, and to save human life.

It is of no use to deny that, to the simply practical man, there is a greater charm in dealing heroically with the acute stages of disease, and, as it were, confronting death face to face, so that the victor's wreath can be unhesitatingly claimed for each success, than in calmly and considerately devising and applying the unexciting measures of protection and prevention which shall secure a far wider, grander, and more enduring—though less easily recognised—success. But, to the man who has cultured his practical faculties with scientific training and research, the greatest charm will lie in finding out the hidden secrets of nature's harvests, and winnowing out from her rich granaries every germ and vestige of disease. This is our special duty in the treatment of such abnormal physiological states as The True First Stage of Bacillary Consumption—

> "A pebble in the streamlet scant
> Has turned the course of many a river;
> A dewdrop on the baby plant
> Has dwarfed the giant oak for ever."

In the diagnosis and treatment of such subtle stages of disease, our *first rule should be "to keep on the safe side"*—the side of precaution. Better place fifty cases under safe precautionary treatment that might *possibly* have righted themselves, than run the chance of leaving one case untreated that is thus allowed to pass beyond the regions of cure. Unfortunately, our greatest obstacles to such a wise course of preventive medicine are raised by the patients, and by the friends who surround them with solicitous yearnings for their good. They think we are "making too much fuss" about what appears to

them to be a comparatively trivial ailment—a little obstinately progressive weakness, loss of weight, and disturbance of temperature. Both urge us to wait for more conspicuous calls for action—that is, till it is too late!

"They dawdle over physics, quackeries, and intended changes of occupation, till the day for cure has passed. Promptitude in this case is everything. I cannot too strongly describe the value of every day wasted—for every day brings nearer, at a rapidly increasing rate, the hour when successful treatment will have lost all its *simplicity*, and become beset with innumerable difficulties."* Whereas, if taken in time the treatment is simple enough, although it must be uncompromising. I have said that our first rule in such treatment should be "to keep on the safe side." We have to decide what to do for the best "in the present state of our knowledge," which is admitted to be incomplete (see pp. 2, 83.) We must, therefore, make our scheme of treatment wide enough in its scope to include the possibility that either of several hypothetical explanations, of the nature and cause of the patient's "Abnormal Physiological State,"† is the true one.

In order to be safe that we are doing our best for the patient, we must make the scope of our treatment include the possibility that his state may be either *ante*-bacillary or *post*-bacillary. It must be admitted that this margin appears to give more than "room and verge

* *Op. cit.*, "On the True First Stage," 1867.

† See "Lectures on the Germs and Vestiges of Disease, and on the Prevention of the Invasion and Fatality of Disease." Reprinted in Part II. of the Author's work "On Diet and Regimen," etc., Seventh Edition.

enough" to be satisfactory; but this cannot be helped, if we would be safe, until our knowledge has had time enough to arrive at greater precision. Of one thing, however, we may be certain, viz., that, if the condition we have to deal with is still *ante*-bacillary, the bacillary stage is so imminent and its advent so insidious that our treatment ought to include the possibility that both conditions exist. We shall do no harm by fighting against the bacillus, even though it be not there, so that this treatment is only supplementary to that which is directed to keeping it out, *i.e.*, to curing the state of predisposition, which invites its domiciliation (see "Habitat," p. 48; and "Mordant-like Conditions," pp. 21 and 22).

But, unfortunately, as I have pointed out (pp. 82, 83), although we may not have been able to discover the bacillus, nevertheless, it may be there in some secret hiding-place, and our search must be renewed from time to time, so as to catch the first demonstrable evidence of its presence. (See p. 84.)

In order to make our treatment as comprehensively safe as I have suggested, it is necessary to bear in mind the principal *portals* by which the bacillus may enter the organisms, viz., the alimentary, the respiratory, the cutaneous, and the generative tracts (see pp. 23—33 and 40). And, in reference to these portals, we must also remember that, in these earliest stages of disease of which we are treating—the bacillus, although not yet domiciled in the tissues, may be on their threshold, sustaining itself on the secretions to be found at these portals preparatory to its advance into the tissues (see pp. 45, 58, 81). "Having regard to the conditions of growth of the tubercle bacillus, it seems likely that the muco-purulent secretion is a peculiarly good medium for its cultivation."

(Dr. Philip, *op. cit.*) Therefore we must lose no time in purifying all these portals by bactericides, and encouraging them by the best remedies at our command to clear themselves of their secretions and throw them out of the organism. Secretion and excretion must be stimulated at all the emunctories.

"The stomach, liver, intestines, and kidneys must be unloaded of their contents, and their secreting glands and cells freed from accumulations, and stimulated to fresh action; this is especially necessary with the liver and mucous glands of the intestines. The portal system of veins must be thoroughly relieved from congestion and obstruction, so that absorption may be accelerated. The skin must be stimulated to action. The pulmonary circulation and the right heart must be freed from impediments; respiration must be set vigorously to work; and the diet must be regulated. In fact, every function of the body must be looked to and called to its account; and, above all, everything must be done promptly"* and *with a jealous regard for the strength of the patient.* Such remedies must always be selected as combine restorative with eliminative properties. Much knowledge, skill and tact must be used in making this selection, and in adapting it to the specialities of each case. It cannot be done on any "rough and ready" lines.

Both during and after this general purification and elimination, we must take every possible precaution to keep the patient out of reach of the enemy. We must never forget that if the bacilli are not already there, the conditions exist which invite their domiciliation—which determine their habitat, if by chance they should be brought within any of the portals. (See pp. 48, 59, 76.)

* "On the True First Stage," 1867, *op. cit.*

In the light of our present knowledge, as set forth in this treatise, we must regard as nothing less than criminal the neglect, which is familiar to every medical man—the astonishing neglect, as we must now feel it to be—to remove the patient threatened with consumption from *dangerous surroundings*. There can no longer be any excuse for the doctor who does not impress this upon the friends of the patient, or for the friends who persist in the neglect after it is pointed out. It is appalling to remember how often we have seen one member after another—in consumptive families—told off to nurse the one dying of phthisis, each falling in turn, as soldiers succeed one another in the breach under the killing fire of the enemy. It has hitherto been looked upon as heroism in the victims. It must henceforward be regarded as murderous on the part of those by whom it is permitted. Those only should be allowed to be most intimately associated with a patient in whom bacilli are known to exist, who have no special susceptibility to its domiciliation.

The practical question at once arises: What are we to do to establish these essential precautions for keeping out of reach of the enemy till the conditions of receptivity have passed?

Every case must stand upon its own merits, and *common sense* should be the best guide as to how to accommodate special circumstances to special cases. But, unfortunately, common sense has always been the rarest of human possessions, and it gets rarer and rarer every day. The curse of the present generation is WANT OF THOUGHT—the hurry to *do* something—instead of first thinking what is best to do. "Pause, and think before you *speak*," was a favourite school-boy lesson of

the past. "Pause and think before you *act*," should be the daily lesson of the present day, for both the young and the old.

If common sense were allowed to rule in the present matter, we certainly should not send patients, specially susceptible to bacillary infection, to private or public institutions where people are allowed to congregate who are suffering from bacillary tuberculisation—to breathe the air polluted by the breaths and sputa from lungs teeming with bacilli and their spores. We should not allow predisposed children to be tended by tuberculous maids, or suckled by tuberculous nurses. (See pp. 26—35.)

If common sense were allowed to rule, we should not let persons, specially susceptible to bacillary infection, drink unboiled milk without first having made sure that it did not come from an udder affected with bacillary mammitis; and we should not let such persons eat at tables supplied with tinned meats, the history of which is only known to those who have thus disposed of their tuberculous cattle.*

* "The precaution of having all milk boiled before consumption will, there is good reason to believe, insure absolute safety against all the ordinary contagions of which milk may be the carrier; and, as we do not consume other kinds of animal food without cookery, there is nothing remarkable in cooking milk as well as flesh. There is, moreover, one point of Professor Brown's report with which we are in complete agreement—that, namely, in which he refers to the filthiness of many dairies."—*Times*, Dec. 6, 1888.

"Tuberculosis and Milk.—Although in the light of recent researches into the bacillary nature and communicability of tubercle by direct experiment, the consumption of milk from tuberculous cows cannot but be looked on as fraught with danger, instances in which such a mode of communication can be absolutely demonstrated are, from the circumstances of the case, not frequently met with. The fact that, even in advanced stages of the disease, the

If common sense prevailed we should not let those suffering from bacillary consumption cohabit with those already predisposed to the same disease (see p. 31.)

Knowing as we do that the tissues and organs of the

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bacilli are often not to be detected in the milk, the generally long incubation period, and the probability that the milk supply has been changed or the animals slaughtered before palpable mischief has been done, as well as the frequency of tuberculosis in man arising from other causes, surround with almost insuperable difficulties all observations on the human subject. But an instance has lately come under our notice which admits of no doubt, and consequently deserves record. The owner of a valuable herd of cows, finding that a large proportion of them were tuberculous, so large a proportion indeed as strongly to suggest infection by association in the sheds, withdrew his milk from the market, and used it, unfortunately without boiling, for fattening his pigs, of which he has a large number, and on which he prides himself not less than on his cows. The result has been that *the pigs have, almost without exception, been affected with the disease* to an extent that has necessitated the slaughter of the whole stock. Another point of practical interest is that he has not been able to discover nodules or other indications of localised tubercle in the cows' udders, a condition still held by some to be necessary to render the milk capable of transmitting the disease. It is much to be regretted, too, that the legal definition of disease in the cow, as laid down in the Dairy and Cowshed Orders, does not include tuberculosis, but is limited to cattle plague, pleuropneumonia, and foot and mouth disease. This definition should be made to include tuberculosis and all eruptions of the udder."—*British Medical Journal*, Jan. 5, 1889.

"At the meeting of the Paris Congress on the 26th July, the question as to the dangers to which people are exposed by the use of the flesh and milk of tuberculous animals, and the means to prevent them was discussed. . . . All agreed that the use of the meat, and particularly the milk, of tuberculous animals, should be regarded as dangerous. . . . The Congress voted, in principle, that the flesh of a tuberculous animal should be seized in totality."—*Lancet* report of Congress, Aug. 4, 1888.

Drs. Hubermaas, Gerlach, Albert, Klebs, Bollinger, Stein, Johne, Baug, Toussaint, Chauveau, and many others, besides Koch, concur

young (see pp. 27, 35) are especially unstable and vulnerable in the presence of the bacillus; that their glandular systems are especially apt to break down; that their alimentary and respiratory portals are in special need of protection, and that their cerebro-spinal systems are especially liable to disease;—if common sense were allowed to rule, we should not let the children of all sorts and conditions of men be passed through the same educational mill, at schools where they are fed alike—or left unfed if they are unwilling to eat—exercised alike, exposed alike, worked alike, punished alike, worried alike —leaving it to the rough laws of *natural selection* and the survival of the fittest to kill off the feeble and susceptible from the crowd. If common sense prevailed, men and women would interpose the *selection of intelligence* to prevent the specially susceptible of their progeny from falling under those rough laws of natural selection which decimate the rising generation.

The practice of medicine was once described as the "Practice of Educated Common Sense." Let us pray that it may never lose this high character! The more we see the common sense of the general population obscured by the feverish heat and fickle glamour of the rage for superficial education, the more necessary it becomes that medical men should keep aloof from the dust and hurry of the crowd, and calmly hold and cultivate their common sense as a sacred charge for the guidance

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in stating that if milk from cattle with tuberculous udders is given for any lengthened period, tuberculosis will be developed.

In Copenhagen "The Control Committee of the Milk Supply Company ordered a fortnightly examination by a veterinary surgeon of all cows supplying milk to their company. This examination to include a most careful inspection of tuberculous udders." —Dr. Woodhead, *op. cit.*

of those in whom it is obscured. There are few more difficult fields in which to exercise "educated common sense" than in attempting to guide the public in the management of The True First Stage of Bacillary Consumption; because it needs to be accommodated to a number of concurrent and often conflicting circumstances; and must be based, as I have said, upon a plan wide enough in its scope to embrace the possibility that one of several conditions may exist alone, or that several conditions may co-exist. (See pp. 91, 92.)

This is exactly what it is most difficult to make the majority of persons understand. The "highly educated" narrow-minded conceit of the present day makes people think that they are bound to understand everything, however much it may be beyond their comprehension; and when they find themselves in a position which they cannot understand, instead of trusting to the authority of a wise and instructed counsellor, who has devoted his life to the subject, they rush at the first chance of some simple formula of belief—some striking flag—on which to pin their faith.

In medical matters this is conspicuously exemplified. At one time they "believe in" cod-liver oil, at another in arsenic, at another in panereatic emulsion, at another in koumiss, at another in mutton chops and porter, at another in grapes, at another in open windows, at another in hot inhalation halls, at another in cold sponging, at another in Turkish baths; at one time they "believe in" compressed air, at another in rarefied air, at another in sea voyages, at another in pine woods, at another in altitudes; at one time they "believe in" hot places, at another in cold; at one time in sea coasts, at another in inland continents; at one time in constant exercise, at another in

constant rest, at another in cotton, at another in wool, at another in peat smoke, at another in no smoke; at one time in inhalants, at another in no inhalants; at one time in electricity, at another in massage; sometimes it is "throw physic to the dogs" sometimes it is physic, physic for ever, on which they pin their faith; and so on *ad infinitum*. It is always *all* one thing or *all* another. In the course of a few weeks of consulting practice a physician will be asked by different patients, and sometimes I am sorry to say by their doctors, too, whether he "believes in" this or that of many of the items in this list of incongruous elements of treatment. The inquirers seem to suppose that the doctor can answer "yes" or "no" to the idiotically-conceived questions as glibly as they are asked.

It seems to be out of the reckoning of these questioners that more than one thing can be "believed in" at once; that any reasonable combination of such important elements can be the proper thing to "believe in"; that one or more of these means of treatment may be necessary at one stage of a case, and one or more at another stage; or that none of them can be wisely applied to the case in hand under present conditions.

My usual answer is, "I don't 'believe in' anything in the sense in which you ask the question. Medical treatment is not to be so thought of or administered. It must always comprise a group of ideas as to the nature of the case, and a group of ideas as to the means of treatment. It is never 'all this' or 'all that.'"

It is this superficial haste to have everything put into a single formula, needing no thought or consideration during action—ruining all prospect that wise advice will be either comprehended or acted upon, that

discourages doctors from taking the pains to give it. So patients run hither and thither, first pinning their faith on this flag and then on that, till all chance of being cured is lost.

I warmly agree with Dr. Clifford Allbutt when he says—speaking of the climatic-treatment of consumption in the altitudes (*Lancet*, Oct. 13, 1888):—

"Let us, then, bring the friends always, and the patients generally, to realise that recovery from phthisis, *however incipient*, probably means a very costly and prolonged system of treatment, and, what is more, a steady, clear-eyed, persevering walk on the part of the patients and friends, if success is to be attained. . . . Now, when a man has had it put straight before him what phthisis means, even *in its small beginnings*, he will learn that a serious, an unflinching and vigilant attitude is his one way of safety; and a little home sickness, some sense of tedium, and some love of change, must not be allowed to turn him from his long and arduous course. . . . Once more, I would urge upon all phthisical patients the importance of incessant medical supervision. Apart as I am from practice in alpine health resorts, I may brush aside all scruples, all suspicions of self or class service, in saying this and repeating it. For those medical men who do practise in these health resorts there must be a fear of misconstruction of their motives when constant supervision seems to them more necessary than it may seem to the patient or his friends. But scruples of this kind, honourable as they are to the physicians, must give way to a clear view of the need of such supervision. The patient must be *kept* at his best—at his best of digestion, as well as at his best of pulmonary disorder. His temperature should

again be scheduled whenever any sense of lassitude is felt, and the catarrhal and other varying conditions of the lungs should be systematically recorded. Not only so, but the regular visits of his doctor keep up the patient's serious resolve, strengthen his will, inform his judgment and discipline his habits. And in all this *lies most of the battle!*" (See p. 119.)

These are the wise and considerate words of an unusually wise, experienced, and independent practical physician. Dr. Allbutt has applied them to cases of *phthisis even "in its small beginnings."* They apply to it more and more strongly as we go further and further back, "in its small beginnings," to what I wish to be emphatically recognised as the smallest of all beginnings — its True First Stage, its pre-tubercular stage — its stage of comparative simplicity, and of comparatively easy cure.

If, at a later stage, "his one way of safety," as Dr. Allbutt says, "is an unflinching and vigilant attitude," "a steady, clear-eyed, persevering walk on the part of the patients and friends, if success is to be attained,"— this is still more important when we are dealing with the earliest stage of all; when everything depends upon our success in keeping the patient from sliding on into the next stage while we are endeavouring to eradicate the first. He is often so close upon the confines of this next stage that it may be impossible to prevent its invasion in the time at our disposal. But the immediate detection of its onset may enable us to cut it short by a *prompt and resolute change of treatment.* "Let well alone," is a wretched axiom for the physician. He should always be on the watch to *do better.*

If the disease runs on, such prompt and resolute

changes of treatment may be required for each new phase. (See p. 118.)

But, if the patient is to have the best chances of cure, these changes must be made with a constant recollection of the "main lines" of treatment—treatment founded on a full appreciation of the basic nature of the disease—and it must be subservient to them. It is distressing to see in daily practice how often this is lost sight of—how often a patient, doing well on a wisely-arranged line of curative treatment, is taken off it, and "shunted on to a siding" because of some accidental intercurrent complaint, and is not restored to the main line till it is too late to continue the curative journey with success. But it is not my purpose in this treatise to discuss the treatment of any stage subsequent to the True First Stage.

Supposing, then, that we have taken the patient out of reach of the enemy—that is to say, so far as it is practicable, out of the chances of bacillary infection by any of its usual portals; that we have cleansed these portals with bactericides; that we have "called to their account" all the secreting and excreting functions; that we have put him upon an easily assimilable unirritating diet (see p.108), including carbo-hydrates and pre-pancreatised hydro-carbons (see pp. 23, 75, 104); and that we have removed any accidental inter-current complaints that happened to be present—"let us never forget that when the patient is thus placed in a position of safety, he is scarcely better off than a leaking ship, when by means of constant working at the pumps she has been brought safely into a harbour of refuge. Unless advantage is taken of this temporary security effectually *to mend the leak*, the ship will go down the first time the pumps are

stopped. . . . It is through ignorance or neglect of this vital object of treatment that cases of early consumption, apparently progressing steadily towards recovery, so often relapse just when the improvement ought to have been made permanent, and thus the best chance of a radical cure is lost." ("True First Stage," *op. cit.*, 1867.)

The question is—What next should be done? What are the best means of restoring "the normal physiological state"? What are the best means of setting right that abnormal metabolism and histogenesis which have allowed the formation and accumulation in the blood or in the tissues,—perhaps of MacMunn's respiratory histohæmatin, adrenal hæmochromogen, urohæmatin, enterochlorophyl, hæmatoporphyrin, and other occult products revealed by spectrum analysis;—perhaps of Selmi's ptomaïnes;—perhaps of Gautier's leucomaïnes, alkaloids and extractives;—perhaps of Crookes' meta-elements;—perhaps of disintegrated proteids and crystalloids, and other *débris* of devitalised molecules which supply the "happy hunting grounds" of the bacilli with those special means of sustenance which determine their habitat? (See pp. 42, 48, 55, 60, 79.)

How are we to find means of treatment which shall permanently arrest these earlier degradations of the normal physiological state?

In answer to this question the first ideas which naturally come into the mind are meteorological—the "*Τὰ μετέωρα*" of Aristotle—some change of climatic surroundings which shall radically alter the conditions of existence.

But before discussing or prescribing remedies not obtainable by "all sorts and conditions of men" it is

well to turn to certain valuable medicinal substances, well worthy of consideration, which are within the reach of all. I have taken for granted (p. 102), that hydrocarbons have been at once added to the diet in their most assimilable forms—oleinous fats, like cod-liver oil, to reach the blood by venous absorption, independent of lacteals and lymphatics which may be blocked, overtaxed, or diseased (see pp. 23, 27, 74-76); solid fats (stearin, margarin, and palmatin) emulsified by pancreatisation, to assist them in reaching the blood by the lacteal route in spite of its damaged or obstructed state.*

When we remember that fat is essential to the formation of healthy cells; that there is reason to believe that the devitalised molecular *débris*, of which we have spoken, are taken up and metamorphosed by cells; and that there may be a "battle of the cells" to resist the bacilli (pp. 15, 58); we shall understand that there are much deeper reasons for this kind of treatment than appear, *primâ facie*, when we merely think of supplying fat to stop waste. I have also taken for granted that carbohydrates (farinaceous foods) have been at once added to the diet—to supply materials for heat and mechanical

* The practical experience of a quarter of a century, quite independent of any hypotheses as to the nature and cause of tuberculosis, has yearly added accumulated evidence from all parts of the world of the value of "Pancreatised, Predigested or Peptonised" foods originated by the Author in the form of Pancreatic Emulsion in 1863. In addition to its value in consumption and in tabes mesenterica, the *British Medical Journal*, August 15, 1885, speaking of predigested food, said, "It has proved useful in many hands, in uræmic vomiting, gastric catarrh, pernicious anæmia, gastric ulcer, and in pyloric and intestinal obstructions. Its introduction has probably done more than any other therapeutic measure of recent times to lessen infant mortality." See also a Leader on "Pre-digested Food" in *The Chemist and Druggist*, January 14, 1888.

force, and thus to economise the fats. For the same reason it may often be necessary to add Alcohol, when there is urgent need to *save time* by supplying materials for rapid oxidation until the organism is charged with materials for tissue formation; but in whatever form alcohol is given, it should always be associated and supplemented with more permanent and substantial forms of food.*

Next in importance, and in the order of therapeutic sequence, come iodoform, chloride of calcium, arsenic—and many would add the hypophosphites; but as to these, although they have a certain usefulness, my own experience is disappointing. I believe that much valuable time is often wasted by too great a dependence upon them, and especially upon certain popular preparations which, while bearing their name, contain them in ridiculously small quantities. But iodoform is a potent remedy, although its most important place is in cases of advanced tuberculisation and disintegration, where the sputa are unavoidably swallowed in considerable quantities. It is also of much value in the earlier stages. When there is a suspicion that bacilli may possibly be lurking in the secretions, ready for an advance into the tissues, or that they may have gained admission to the blood, iodoform should always be administered as a precautionary measure. Its value was first brought to my notice by Dr. Stiles Kennedy, of Newark, Delaware, in a brochure which he sent me when I was editing my Reports on Medicine in 1869. Speaking of his own experience he said, "Any medicine that will assuage the sufferings of the phthisical patient is indeed a boon, but iodoform

* See the Author's opinions on Alcohol, pp. 7, 8, "Diet and Regimen," *op. cit.*, Seventh Edition.

offers more; there is every reason to believe that it exerts a most potent influence in discussing and removing the disease in its early stages. . . . I do not know that iodoform has been used in any case of open scrofula, *i.e.*, ulceration of the lymphatic glands; but several cases bearing that diathesis, and springing from that cause alone, so far as could be judged by the senses, have been treated successfully." But we owe the present *revival* of the remedy in consumption to the careful experiments and reports of Dr. Shingleton Smith, of Clifton. There is very seldom any difficulty in getting it taken and well borne if prescribed in a pill —which I contrived after reading Dr. Kennedy's paper in 1869—containing gr. ii. iodoform, and made up with sugar of milk and glycerine of tragacanth. This should be varnished and scented with coumarin or coffee, and taken twice or thrice a day *half way through a meal.* If it causes any sensations of nervous perturbation, which is rarely the case, the dose should be diminished, and if there is diarrhœa or sickness, it should be discontinued till they are allayed, and during this interval, sulpho-carbolate of soda (a valuable remedy which we owe to Dr. Sansom) should be given instead.

The next remedy of importance is chloride of calcium. It deserves to be considered a curative medicine; not from any direct action upon bacilli or their food, but from its specific action upon lymphatic glands. Its action in relieving and reducing enlargement of superficial lymphatic glands is well known; and there is every reason to believe that it is no less potent in its action upon adenoid tissue generally, and upon the deeper glands, especially the mesenteric system, to which it is more directly applied through the alimentary portal.

It should be given in doses of ten to fifteen grains twice or thrice a day, well diluted, and it may be advantageously combined with bark or quinine. With our present knowledge of the connection between bacillary consumption and affections of the lacteal and mesenteric system, we cannot be surprised that clinical experience has proved the value of chloride of calcium in the earliest stages of consumption, and that this is especially the case in early life. (Pp. 26, 27, 73.)

Arseniate of soda comes next in importance, especially if loss of flesh is rapid. I think it must be regarded rather as a "skid" to "brake" the wheels of a descending vitality while restorative measures are being applied, than as a strictly curative remedy; but as a tonic, antiseptic and anti-dyspeptic, it does much to help the cure in a large number of cases—when tuberculisation is imminent. But it is most valuable in developed phthisis after tuberculisation has occurred. One of the best means of administering it is in the form of La Bourboule water, mixed with one-third of boiling milk.

CHAPTER VII.

Diet and Regimen continued—Strong meat belongeth to them that are of full age—Important assistance by Pancreatine, Pepsine, Malt Extract—Clothing by day and night—Dangers of Catarrh in relation to Bacilli—Warming and ventilation of rooms—Protection from draughts, etc.—Baths and ablutions—Exercise: How to estimate the proper amount for the weak and the strong—Maclaren's observations on physical education—Over-fatigue singularly disastrous to the consumptive—Explanation of its appalling effects—The fever of exhaustion—Necessary precautions—Walking, riding, cycling, massage—Observations of Angelo Mosso, Aitkin, Brown, Brunton Williams—Proposed riding and cycling tours round United Kingdom, Advantages for both sexes—In prescribing climatic treatment each case must be specially considered—Dr. Loumiss, of New York, and the Adirondack Mountains—The Swiss Alps—The Rocky Mountains and New Mexico—South Africa—New Zealand—English health resorts—Exceptional management of the very young.

BEFORE leaving the subject of food and medicine, I must insist upon the great importance of keeping up the habit of taking regular solid meals of the old-fashioned family type; consisting of a fair admixture of fish, meat, poultry, game, eggs, vegetables, puddings, bread, and fruit, with a moderate quantity of beer or wine, and of coffee, tea, or cocoa, taken at regular intervals of three to five hours,—the intervals being shorter in the first half of the day and longer in the second half. When it has been necessary—as it often must be when cases first come under treatment (pp. 102, 104)—to adopt smaller, more fluid, and more frequent meals, we should

never forget that "strong meat belongeth to them that are of full age," and that the stomach very readily loses the *habit* of taking and disposing of "strong meat"—if it is kept too long and too frequently supplied with food that does not call upon the digestive apparatus for the full performance of its functions.* Therefore we should always keep in view, as an important feature of dietetic management, the return to solid and less frequent meals, as soon as it is possible to do so with safety. This possibility may often be hastened by giving pancreatine, pepsine, and diastase, either with or soon after meals, to assist and accelerate digestion and assimilation.† I may mention here—as it does not seem to be always understood even now—that when pancreatine is given it should be the POWDERED PANCREATINE, as none of the fluid pancreatines have any considerable power of *emulsifying fat,* and that as this preparation has the properties of pepsine and of diastase—as well as the emulsifying power—there is no need to give malt extracts as well as pancreatine, and that pepsine is only needed when the power of the pancreatine to digest albuminoids requires to be supplemented.‡

The importance of wool-clothing next the skin, both night and day, and of so regulating the clothing as to protect from chill and cold, but not to oppress with its weight, or to keep the body perspiring when at rest, are

* See some excellent papers by Dr. D. J. Brakenridge, of Edinburgh, "On the Influence of a Digestive Habit."—*Medical Times and Gazette*, June, 1868.

† See Appendix for rules for the Construction of Normal Diets and illustrative forms.

‡ See the Author's Paper on the "Special Action of the Pancreas on Fat and Starch." Proceedings of the Royal Society, No. 97, 1868.

so well known now that I need not dwell upon them here, further than to add that "during sleep it is equally important to keep the body pleasantly warm, and to avoid keeping it over-heated; and that as serious changes in the weather may happen in the night, and the lowest temperature in the twenty-four hours naturally occurs between two and six o'clock a.m.—the ventilation and clothing must be prepared for these contingencies. Some extra article of clothing should always be at hand to put on the bed if necessary. . . . It is most important to keep the arms warm both day and night. Cold arms act as refrigerators to the blood immediately before it is discharged into the heart and lungs. . . . No sitting or sleeping-room should be left long without a fire; and every room in which persons live, either by day or by night, should have some opening by which it communicates with the outer air; but this should be so arranged that no draught can fall upon the persons in the room. If several rooms are occupied by turns during the twenty-four hours, the temperature of any one should not differ greatly from that of the rest. *No draught should blow upon a bed*, and during sleep the whole body should have one covering at least of woollen material; for while it is very important to keep the air of sleeping rooms fresh, it must be remembered that the body is more susceptible of chills during sleeping than waking, and that changes of temperature in the outer air are especially apt to occur during the night, and are therefore in danger of producing chills before they are observed." *

Recent observations have proved that the catarrhal secretions of mucous membranes form tempting hiding-

* "On Diet and Regimen," *op. cit.*, Seventh Edition.

places for bacilli while waiting for the chance of more dangerous domiciles in the tissues; and this should be a warning to those who, in their zeal for open windows and open carriages, set at defiance the experience of the past as to the dangers of thus giving colds to those constitutionally disposed to catarrh. (See pp. 43, 68, 92.)

Great follies are committed in the matter of the daily bath, some going to one extreme and some to another. As it is of great importance to adopt a plan by which the body may be invigorated without the danger of inducing chills and catarrhs, which are so dangerous to the consumptive, I should like to repeat here the directions given with this view in my work on "Diet and Regimen":—"Warm baths, Turkish baths, vapour baths, shower baths, cold plunges, and sea baths should only be used under special medical orders. During ordinary health the skin of the body and limbs should be smartly rubbed once in twenty-four hours, first with a rough towel (or large sponge) wet with cold water, and then with a dry towel till in a glow. The bather should stand on a dry rug while using this '*cold friction bath*,' and it should not last more than one or two minutes, including both the wet and dry rub. Salt may be advantageously added to the water, and the bath may be used either on rising or going to bed, according to the feelings and convenience of each individual, the morning being preferable as a rule.

"When water cannot be borne cold, it must not be used tepid, but *scalding hot*. The momentary application of scalding hot water to the skin, immediately followed by a brisk friction ('hot friction bath') will produce a *direct action glow* much like the *reaction glow* following the application of cold.

"If the weather is cold, or the person delicate and chilly, the upper half of the body should be uncovered and bathed and rubbed first, and then a woollen vest should be put on, and the lower half uncovered and bathed and rubbed. . . . Except under definite medical orders, it is not advisable to sit or stand in cold water."

On the important subject of exercise, the foolish extremes advised for patients are even more dangerous than in the case of baths. It is necessary, therefore, to give a few plain directions under this head. "During ordinary health, some part of every day ought to be spent out of doors; and in ill health it is of great importance not to discontinue the observance of this rule without good reason, for although in certain states of disease it may be very important to remain in-doors, it must not be forgotten that proper clothing, goloshes, respirators, waterproofs, and umbrellas, may make it not only safe but advantageous to go out of doors for exercise, when without these it would be very injurious. Out of door exercise should be as active as the strength will allow, and should always be continued *up to the point of slight, but not over-fatigue.* This will be the best measure of the proper amount for both the weak and the strong. (See p. 115.) Unless the air is pure and the person strong, exercise before breakfast is more likely to do harm than good." Mr. Maclaren has well observed in his excellent work on "Physical Education,"* that "a most important principle in exercise, and one that should ever be borne in mind, is that it should be regulated by *individual fitness;* for the exercise that scarcely amounts

* "A System of Physical Education." By Archibald Maclaren, of the Oxford Gymnasium.

to exertion in one person, will be injurious and dangerous to another; and not only is this inequality observable among different individuals, but the same individual may have parts of his body possessing special power or presenting special weakness. A man may have limbs capable of transporting him at the rate of four miles an hour, throughout the day and for many days in succession, but the heart or lungs all unequal to the effort; or he may have an organisation so fine and a temperament so susceptible to stimulation or excitement that the one is an abiding danger to the other."

Over-fatigue, by which I especially mean *the effects of too protracted exercise*, is singularly disastrous in consumption in all its stages. In the early stage I have often seen a patient—who was going on so well as to believe he might indulge in as much exercise as he pleased—make a disastrous descent, from which he never recovered, after *foolishly prolonged fatiguing exercise*. And no form of exercise is worse in this respect than walking.

These cases are so appalling that I have often endeavoured, in vain, to find any adequate physiological explanation of such a tremendous downfall from a cause that, *primâ facie*, ought at most to have been temporary in its effects. But I think I can now explain the matter.

Suppose the patient to have a few bacilli domiciled in his system—the propagation and advance of which is being successfully kept back by the organismal defences, fortified by the influence of judiciously directed diet, regimen, and other medical treatment. From what we have already fully discussed, we may assume that this improved state signifies that, while maintaining his formative and reparative powers, he is *starving out* the bacilli, as it were, by keeping his organisation clear of

those *débris* of molecular disintegration, etc. (see pp. 15, 39, 42, 48, 49, 58, 63-68, 73, 74 103), which constitute their sustenance. Now, if, under these circumstances, he not only exhausts his formative and reparative powers and calls upon them for unusual activity while so exhausted, but *floods his system with the materials which supply sustenance to the bacilli*, and at the same time raises his temperature a few degrees by fever; he establishes in special force the two first laws which dominate habitat (see p. 48), viz., the presence of appropriate food and the presence of appropriate temperature—which are at once taken advantage of by the bacilli to multiply their host and overwhelm the patient.

That over fatigue is competent to produce these effects has been practically demonstrated in the too protracted marches of armies, and by experiments. "Normal health comes to be conditional on an incessant formation, transformation, and elimination of the effete or old organic materials, which must give place to the new. It is this effete material (in whatsoever form it is found) which, therefore, represents a series of partial deaths; and which, as the result of organic functional operations, constitutes life, during which the tissues and organs in the processes of their metabolic changes, perform a constant function of disintegration—fabricating during these processes those 'alkaloids' and extractives—'those *x*, *y*, *z*'s of pathology' which must be regarded as veritable scoria (Dr. A. M. Brown), or physiological ashes (Dr. Lauder Brunton) resulting from the processes of combusion of the elements of organic tissues. . . . [See p. 66.] Their accumulation may take place under two widely different conditions: as when there is an excess of 'extractive matters' and 'alkaloids,'

with normal but inadequate elimination by the emunctories: or, the production of delcterious materials being normal, their elimination is inadequate from disease or derangement of the emunctories. Hence, auto-infection may result from excessive production, and inefficient (*i.e.*, inadequate) elimination, the emunctories remaining sound, *a condition which is constantly seen in all forms of physical over-taxation or over-exertion*, as in a prolonged march, or by excessive drill, especially in young and adolescent soldiers. Of such examples army medical officers acquire considerable experience.

"In a paper recently submitted to the '*Academia dei Lineei*' (to be printed in its Transactions) the physiology of fatigue has been carefully worked out by Professor Angelo Mosso, of Turin, with a view to the determination of the pathological manifestations which accompany that physical condition. He has found that when fatigue is carried *beyond the moderate stage* at which it is decidedly beneficial [see p. 112], the blood is subjected to a decomposing process through the infiltration into it of substances which act as poisons—substances which, when injected into the circulation of healthy animals induce malaise, and all the signs of excessive exhaustion. It was on the soldiers of the Italian army that Mosso's experiments were made. . . . This '*fever of prostration*' engendered by over-exertion is a very characteristic one, due to the proteid embarrassment which results from the functional disturbance of the tissues. The changes which take place (chemical and functional), although they cannot be actually seen, yet to some extent they can be measured, judged of and made out, or realised from their effects." (Sir W. Aitkin, *op. cit.*)

Some kinds of exercise are to be greatly preferred to

others for consumptive patients. For a variety of reasons, and especially on account of economy, exercise is too apt to take the form of walking, which is by no means the most desirable for delicate persons; it cannot compare with horse-riding. I pointed this out in my work "On the Medical Aspects of Bournemouth":—"In speaking of air and exercise I wish to impress the importance of horse, pony, or donkey riding (in proper weather, according to the strength, etc., of the patients). . . . Neither walking nor driving can compare with riding for consumptive invalids. It has even been regarded by very eminent authorities as a 'cure' for incipient consumption. . . . Consumptive patients are often accompanied (to health resorts) by relatives who have the same hereditary and constitutional tendencies as the patients themselves, and who, frequently, are only one or two stages behind the so-called 'patient' in the progress of the same family scourge. To these persons horse-riding and pony-riding would be most potent aids to developing in their favour the curative influences of climate. . . . I am endeavouring to induce the riding establishments at Bournemouth to introduce troops of sturdy and quiet ponies trained for invalids to ride."

I am glad to find that Dr. Theodore Williams agrees with me in this. "Riding exercise from the time of Sydenham, has been generally acknowledged to be peculiarly beneficial to consumptive patients who are strong enough to bear it, and it is difficult," he says, "to find a form of exercise which so admirably answers the purpose of giving plenty of fresh air and thoroughly warming both body and extremities with so small an amount of fatigue. Bicycling and tricycling combine

the advantage of fresh air in abundance and a great amount of exercise chiefly of the legs, but there is no doubt that the whole muscular system is brought into play, though often too strongly. In cases of weakness, and great loss of appetite and flesh, when at the same time, the tubercular disease is not very active, 'Massage' of the muscles of the body after the Weir-Mitchell method can be practised with considerable benefit and with decided gain in weight. Under its influence appetite and digestion improve, and the circulation becomes more vigorous, colour returns, and the quantity of food consumed is sometimes astonishing." *

When The True First Stage of Bacillary Consumption is first detected, and while preparations are being made for change of occupation and surroundings, I have found Massage of great use. It should be moderate in severity and always *centripetal* in direction. In these cases, as we have seen, the ptomaïnes, leucomaïnes, alkaloids, extractives, myohæmatins, *débris* of molecular disintegration, etc., have been allowed to accumulate in the tissues and organs. It is probable that *centripetal* massage drives them into the current of the circulation and facilitates their destruction or excretion. Hence two or three weeks of skilled massage is an excellent prelude to out-of-door exercise.

With regard to cycling, I do not think these patients should be allowed bicycling; and if tricycling is allowed it should be on a *double tricycle* accompanied by a friend stronger than the patient—who can take the stress of the work if the patient is fatigued, push the tricycle up hill for him, and protect him from accidental bad weather, and, if on a tour, regulate the amount of his daily

* "On Pulmonary Consumption," *op. cit.*

exercise according to circumstances, and superintend his hotel and other management; otherwise the risks of cycling are apt to be greater than the advantages. This caution also applies to *tours* on horseback; they should not be made alone.

Some years ago I sketched out a scheme for a riding tour round the United Kingdom—varied, at special places and seasons, by boating and sailing excursions—bringing the tourist to the various districts at their most favourable seasons, and arranging halting places for rest and for medical examinations at stated intervals. I submitted the plan to Messrs. Cook, the tourists, and tried to induce them to organise parties provided with tickets, etc., to secure proper supervision and accommodation, as in their holiday tours abroad. But at that time they did not think it would pay, and the matter was dropped. For a large number of the most incipient cases such tours would answer every purpose of change of scene, change of air, out-of-door exercise, etc., with these great advantages:—that they could be started on very short notice, that the tourist would always be within reach of home and of friends in case of need, would be secure of reliable medical aid in case of intercurrent illness, and of periodic medical examination—to see whether he was still on the right track, and if not to alter it (see p. 101)—and he would be able to get that very important desideratum, *wholesome English fare*, and to avoid, that apparently inevitable accompaniment of foreign travel, the risk of being poisoned by bad water and stinks.

Such tours would also have the very important recommendation—that they would be available for girls and women, who, while unhappily more liable to consump-

tion than men, are, by numerous inevitable difficulties, unable to undertake the more enterprising schemes of climatic change which are open to men. Fortunately, these disabilities are being every day reduced in number; and even the journey from London to the Adirondack Mountains has been made comparatively easy. The Atlantic voyage is now almost a holiday trip, and the extension of the Chateangay Railroad enables the traveller to leave New York by a night express, to breakfast at Plattsburgh, and reach Saranac by noon next day without further change. But I advise any patient going on this excursion to stay in New York long enough to consult Dr. Loumiss—who has done so much to develop the Adirondack cure,—and take full instructions from him as to the management of "life in the mountains."

Nothing in the way of travel can be more suited for the treatment of The True First Stage of Bacillary Consumption than the tour of the Rocky Mountains,* the Adirondacks, the Swiss Alps, the high grounds of South Africa, the northern parts of New Zealand, and the like. The questions of more land or more sea, more heat or more cold, higher altitudes or lower altitudes, must be settled after a full consideration of the specialities of each case. (See pp. 94, 103, and Dr. Clifford Allbutt's remarks, p. 100.)

However advantageous it may be *in the later stages* of developed phthisis to settle patients for a season at some selected health resort, this is not the best thing for the majority of persons *in the true first stage*. Except

* For full details of "New Mexico and the Rockies," see Chap. IX. of "The Medical Aspects of Bournemouth," Second Edition, *op. cit.*

in the case of children, who can find sufficiently invigorating and amusing occupations within a limited area, and who, as a rule, should be exposed to as little travelling as possible.*

It is comparatively easy to surround the very young with all the conditions of existence essential to health, without going far-afield if people will only be guided by common sense (see p. 97.) "Happily the day has passed when it was believed that there must be a *specific climatic cure* for every disease. No professional or popular delusion has led to the sacrifice of more lives, or to more domestic disruptions, blasted hopes, and weary disappointed longings for that which, while impossible was still believed to exist. The conviction of to-day that even the most typical climate is only a more or less powerful adjuvant to other medical treatment—a more or less favourable condition of existence—brings us within the regions of reason and common sense. . . . It easily occurs to the mind of the invalid that, by rushing after the region of lowest mortality for each season, he may defeat death. But too often this idea leads to almost as mad a dance as that of a moth around a candle." †

In prescribing the exact form which the change in the surroundings and in the manner of living is to assume, we must always take into account the temperament and possibilities of the individual, and also any strong longings for this or that, the indulgence of which may give the chief stimulus to restorative treatment.

"Let him who, while yet young and in health, only fears the future inroads of some constitutional or here-

* See the Author's article, "Bournemouth for Children," in the *Prov. Med. Journal*, Feb., 1889.

† "The Medical Aspects of Bournemouth," *op. cit.*

ditary taint, and whose temperament rebels against the trammels of a cautious life at home,—let him, if he has no ties or responsibilities to prevent, take his life in his hands and his knapsack on his back, and go forth into this beautiful world of land and sea, mountain and river, valley, forest, and plain, to explore the grand battlefields of nature, and *run the risks* which such explorations inevitably incur, for the sake of the great harvest which they sometimes bring—saying to himself, 'Nothing was ever done without risk, and nothing great without danger, therefore I have taken my determination.' To shut up such spirits for months in Davos, Bournemouth, the Riviera, Arcachon or any other health station in the world, is like attempting to keep swallows in a cage" (*op. cit.*).

But such stations may with advantage be utilised as resting places during certain phases of a health tour, as centres to and from which to travel, and especially as harbours of refuge during temporary illness or stress of weather, where domestic comforts and good medical advice can be secured. (See p. 118.)

For these and many other reasons it is advisable that patients who have passed the True First Stage — in whom tuberculisation is advancing, or who are in still later stages of phthisis—should be quietly settled in judiciously selected health stations.

But it is not for such cases that I have written this treatise. My object has been to point the way by which such fates may be avoided, by nipping "in the bud" the "worm" that would have fed upon the "damask cheek."

THE END.

APPENDIX.

I.—ESSENTIALS OF A NORMAL DIET.

A HEALTHY adult man of average stature, taking moderate exercise, will require and can consume, daily, from 32 to 40 ounces avoirdupois of *dry* nutritious food, which should have the following characters :—

1. About $\frac{1}{20}$ must be mineral matter.

2. From $\frac{2}{5}$ to $\frac{1}{2}$ may be water, leaving $\frac{3}{5}$—or not less than $\frac{1}{2}$—or from 15 to 20 ounces of anhydrous solid alimentary material.

3. Three or four ounces of plastic matter must be combined with three or four times that quantity of heat-giving material.

4. The heat-giving constituents must contain a mixture of fats (hydro-carbons) with saccharine materials (carbo-hydrates), in the proportion of about 1 of the former to three of the latter.

5. These heat-giving constituents should supply from 6 to 10 ounces of carbon, the exact amount required varying with season, exercise, etc.

6. The Articles of Food must be sufficiently varied to meet the requirements of the taste and of the appetite, and their Mechanical and other Conditions must be suited to the digestive powers of the stomach.

In addition to these characters, every complete diet must contain some potash-vegetable or fruit ; and the total amount of water taken in 24 hours, *including that contained in the dry food*, must not be less than 70 ounces avoirdupois.

II.—DIET TABLES

FOR THE MAINTENANCE OF HEALTH IN ADULTS LIVING IN THE CLIMATE OF THE UNITED KINGDOM.

Each diet table contains all the essential elements of nutrition, in forms, quantities and proportions necessary to the maintenance of health. The tables have been made complete without Alcohol, leaving this to be ordered or not, according to circumstances. But, if fermented liquors are added to any diet table, the quantity of carbon which they contain must be borne in mind.

It will be observed that the totals of the corresponding columns of the analyses are nearly the same in all the diet tables, showing the important fact that all the essentials of a normal diet may be equally secured in a diet that is simple and cheap and in one that is complicated and expensive.

In all diets Salt must be used, and in those which do not include Potatoes, some other Potash-vegetable, or Fruit, or Cresses, or Lime or Lemon juice must be taken. (See Table V.)

No. 1.

Liquid. Fluid Ozs.	Dry. Ozs.	Food for 24 hours.	Water. Ozs.	Plastic. Ozs.	Fat. Ozs.	Saccharine. Ozs.	Carbon. Ozs.
	6	Meat, Poultry or Game (cooked)	3·97	1·35	·53	—	·42
	4	Fish	3·19	·66	·03	—	·02
	10	Bread	4·20	1·00	·07	4·53	1·93
	8	Potatoes	5·81	·14	—	1·84	·76
	2	Rice	·18	·10	·01	1·63	·73
	2½	Sugar	—	—	—	2·50	1·05
	2½	Butter	—	—	2·50	—	1·85
5		Milk	4·34	·25	·17	·21	·22
16		Coffee	15·77	—	—	·22	·10
16		Tea	15·05	—	—	·05	·02
17		Water	17·00	—	—	—	—
54	36		70·41	3.50	3·31	10·98	7·01
		In Plastic matter					1·89
		Total					8·99

No. 2.

Liquid. Fluid Ozs.	Dry. Ozs.	Food for 24 hours.	Water. Ozs.	Plastic. Ozs.	Fat. Ozs.	Saccharine. Ozs.	Carbon. Ozs.
	8	Meat, Poultry or Game (cooked)	5·29	1·80	·71	—	·56
	12	Bread	5·04	1·20	·08	5·44	2·32
	12	Potatoes	8·71	·20	—	2·76	1·14
	2	Butter	—	—	2·00	—	1·48
	2	Sugar	—	—	—	2·00	·84
5		Milk	4·34	·25	·18	·21	·23
16		Chocolate	15·20	·07	·32	·38	·42
16		Tea	15·95	—	—	·05	·02
16		Water	16·00	—	—	—	—
53	36		70·53	3·52	3·29	10·84	7·01
		In Plastic matter					1·90
		Total					8·91

No. 3.

Liquid. Fluid Ozs.	Dry. Ozs.	Food for 24 hours.	Water. Ozs.	Plastic. Ozs.	Fat. Ozs.	Saccharine. Ozs.	Carbon. Ozs.
	18	Bread	7·56	1·80	·12	8·15	3·47
	3½	Cheese	1·28	1·07	·89	·08	·70
	3	Bacon	·76	·25	1·88	—	1·48
	1½	Sugar	—	—	—	1·50	·63
5		Milk	4.33	·25	·17	·21	·22
20		Chocolate	19·00	·10	·40	·50	·50
20		Tea	19·93	—	—	·07	·04
20		Water	20·00	—	—	—	—
65	26		72·86	3·47	3·55	10·51	7·04
		In Plastic matter					1·87
		Total					8·91

No. 4.

Liquid. Fluid Ozs.	Dry. Ozs.	Food for 24 hours.	Water. Ozs.	Plastic. Ozs.	Fat. Ozs.	Saccharine. Ozs.	Carbon. Ozs.
	8	Fish	6·38	1·33	·06	—	·05
	16	Bread	6·72	1·60	·11	7·25	3·09
	8	Potatoes	5·81	·14	—	1·84	·76
	2½	Butter	—	—	2·50	—	1·85
10		Milk	6·93	·40	·28	·34	·36
20		Cocoa	19·10	·10	·60	·20	·56
	1½	Sugar	—	—	—	1·50	·63
25		Water	25·00	—	—	—	—
55	36		69·94	3·57	3·55	11·13	7·30
		In Plastic matter					1·93
		Total					9·23

No. 5.

Liquid. Fluid Ozs.	Dry. Ozs.	Food for 24 hours.	Water. Ozs.	Plastic. Ozs.	Fat. Ozs.	Saccharine. Ozs.	Carbon. Ozs.
	16	Bread	6·72	1·60	·11	7·25	3·09
	3	Peas	·41	·65	·04	1·40	·65
	4	Bacon	1·14	·33	2·50	—	1·98
	2	Cheese	·73	·61	·51	·05	·40
10		Milk	6·94	·40	·28	·34	·36
20		Coffee	19·71	—	—	·28	·13
	1	Sugar	—	—	—	1·00	·42
35		Water	35·00	—	—	—	—
63	26		70·65	3·59	3·44	10·32	7·03
		In Plastic matter					1·94
		Total					8·97

No. 6.

Liquid. Fluid Ozs.	Dry. Ozs.	Food for 24 hours.	Water. Ozs.	Plastic. Ozs.	Fat. Ozs.	Saccharine. Ozs.	Carbon. Ozs.
40		Milk	34·68	2·00	1·40	1·68	1·80
	4	Rice	·36	·20	·02	3·26	1·46
	3	Eggs (two)	2·15	·45	·32	—	·26
	2½	Sugar	—	—	—	2·50	1·05
	1	Butter	—	—	1·00	—	·74
	9	Bread	3·78	·90	·06	4·08	1·74
30		Water	30·00	—	—	—	—
70	19½		70·97	3·55	2·80	11·52	7·05
		In Plastic matter					1·92
		Total					8·97

III.

Dr. Theodore Williams (*op. cit.*) gives the following judicious dietetic directions for consumptives :—"When the appetite is good, and a fair amount of open-air exercise can be taken, a dietary like the subjoined generally results in gain of flesh :—

"8 to 9 a.m. Breakfast.—Bread (whole-meal if possible) and milk (¾ pint), or porridge and milk, rendered a little more digestible by the addition of a little ground malt, or A. B. C. cereals, white wheat, or Durber's wheat or hominy with milk; fried bacon, egg, or fish, or poultry; a cup of coffee or cocoa and bread and butter.

"1 to 2 p.m. Luncheon or Early Dinner.—Plenty of tenderly cooked meat, with potatoes and fresh vegetables; light farinaceous puddings, a little ripe or stewed fruit, and a glass of sound sherry, or a larger one of claret, or ½ pint of bitter ale.

"4 to 5 p.m.—½ pint of milk with a rusk or biscuit.

"7 to 8 p.m. Dinner or Supper.—Plainly-dressed white

fish, to be followed by meat, mutton or beef, alternated, to give variety, with poultry and game, vegetables ; sweets as at luncheon, and a glass of sherry with water, or a larger one of claret, hock, or Chablis, followed by a cup of hot coffee or a glass of hot water, if needed to assist digestion.

"In cases where the appetite is very capricious, as in weakly women, less food can be taken at a time, and more frequent feeding is requisite, so that a dietary something like the following is to be preferred:—

"7 a.m.—½ pint of warm milk with a dessert-spoonful of brandy or rum.

"9 a.m. BREAKFAST.—Milk with cocoa or coffee, bread and butter, bacon, fish, or poultry.

"11.30 a.m.—Eggflip, *i.e.*, one egg beaten up with a dessert-spoonful of brandy ; or ½ pint of milk or a glass of koumiss or kèfir.

"1 to 2 p.m. LUNCHEON.—As in first dietary.

"4 p.m.—Same as at 11.30, or, if desired, a cup of tea with milk and biscuit, or slice of bread and butter.

"7 p.m.—Beef tea with toast and a glass of wine.

"10 p.m.—Some farinaceous food, such as milk, gruel, arrowroot, etc.

"These forms may, and should, be endlessly varied according to the digestive capacity of the individual, provided always that the relative proportions of the food-stuffs are maintained, and the stimulants introduced in amount and kind to assist digestion and prevent waste."

IV.

In my work on "Loss of Weight" (*op. cit.*), I said, in 1880, what I may repeat, after nine years' further experience, "I cannot speak too highly of the following 'SPECIAL NUTRITIVE' (pancreatised milk, egg, and arrowroot, with wine or brandy), as a means of feeding and fattening patients unable to take or to digest a sufficiency of solid food. I have used it very largely in practice (and so have numerous medical

friends), and I have seen emaciated patients grow fat upon this food alone. This need not surprise us, for its analysis shows it to contain all the essentials of a normal diet in proper quantities and proportions in the forms most easy of digestion and assimilation.

"Beat up an egg, both white and yelk, quite smooth and *free from stringy particles;* stir it well into half a pint of hot milk in which enough arrowroot has been boiled to make it about as thick as cream; add a wineglassful of sherry or a tablespoonful of pale brandy, and some fresh nutmeg and sugar; mix all thoroughly by pouring from cup to cup, and when it is *just cool enough* to be borne by the mouth (about 140° Ft.), stir in ten grains of Savory and Moore's powdered pancreatine.—A person living entirely on this diet should drink fresh-made lemonade when thirsty."

V.

The *Special Nutritive* may be advantageously alternated with the following INVALID SOUP, which may be taken cold as a jelly, or warm as a soup. Since I first published the recipe in 1864 it has proved invaluable in a large number of cases. It paves the way for solid food.

"Gravy beef 1 lb., scrag of mutton 1 lb., isinglass 2 oz., vermicelli 3 oz., mushroom ketchup three tablespoonfuls, corns of allspice 24, sage a sprig, cold water three quarts.

"Put the isinglass and the meat, cut small, into the cold water, gradually boil, skim well, and then add the other ingredients; simmer four or five hours till reduced to one quart. Strain through a fine hair sieve, and carefully remove all fat; add salt to the taste. Calfsfoot may be used instead of isinglass when procurable; and when not contra-indicated a little *solution* of cayenne pepper may be added; and the flavour may be varied by the substitution of Worcester or some other wholesome sauce for the ketchup. A full dose of Savory and Moore's powdered pancreatine should be taken directly after the soup.

VI.

The following forms a sort of KOUMISS, and is not only pleasing to many palates, but to stomachs that cannot bear milk in the usual forms:—

New milk well curdled,* ℥viii. Remove half the curds and put the rest of the curds and whey into a wine bottle ; add ℥iij of "Hungarian sparkling" (Max Greger's best quality) briskly effervescing,† shake the whole sharply, and pour it frothing into a tumbler.

See other receipes for the sick room in "Diet and Regimen," 7th Ed. (*op. cit.*).

VII.—BACTERIOLOGY OF SNOW.

"While the bacteriology of ice and hailstones has been studied with considerable success by Drs. Fränkel, Bischoff, Mitchell Prudden, Pumpley, Hills, Stoben, A. V. Poehl, Bordone-Ufreduzzi, Bujwid, etc., that of snow has been, up to the present, almost wholly neglected. Even in Russia the subject has been touched only in a cursory way by Professor A. V. Poehl in a paper on the water-supply of St. Petersburg, in the *Vratch*, Nos. 8 and 9, 1884, p. 119. In it he points out : 1, that snow always contains viable microbes liquefying gelatine : 2, that, when snow falls, the first portions invariably contain greater numbers of bacteria than the subsequent ones (for example, 8,324 per 1 cubic centimètre of snow-water against 3,380 several hours later) ; 3, that, when snow lies on the ground, the superficial layers become richer in microbes (for example, 780 just after the fall, against 962 about three hours later). The fact is of interest from a sanitary point of

* By far the best and easiest way of making nice curds and whey is to use the Curdling Powder sold by "The Sanitary Association, Gloucester," to be obtained by post, with directions for use.

† To keep the wine well "up" for further use, stop the bottle with an elastic cork.

view, as Dr. Poehl's researches furnish an additional proof that exposure of microbes to low temperatures does not destroy their vitality; at least, in certain species of micro-organisms. In many countries, such as Russia or Sweden, snow forms, so to speak, a natural ground or soil during several months of the year, receiving excrementitious matter and every possible kind of refuse and filth. In spring, when the snow melts, it is imbibed by the soil, carrying with it all the polluting matters referred to. Hence an interesting question arises: Are such microbes as happen to be present in these matters in any way changed by their contact with snow, or not? This point can be determined only by further bacterioscopic researches. A contribution to the subject has just been published in the *Vratch*, No. 37, 1888, p. 727, by Dr. F. G. Ianovsky, of Kiev, who has examined bacterioscopically, under Professor K. G. Tritshel's guidance, a February snow in its purest state, collected both immediately and from one to three days after its fall. This observer has found: 1, that even when collected during its fall, snow is invariably found to contain living bacteria in considerable numbers, varying from 34 to 463 per 1 cubic centimètre of snow-water; 2, that their number does not decrease from exposure of snow to low temperatures (—16° C.=3·2 Fahr.) for several days; 3, that the following three species of microbes are met with constantly in great numbers: *a*, a large diplococcus composed of ovoid cocci, endowed with energetic motion, and characterised by its rapidly liquefying jelly; the test-tube culture on the third day, forming greenish colonies along the track of the needle, assumes the shape of a funnel-like sac with a whitish flocculent deposit, while on the fifth the whole medium becomes liquefied, the precipitate sinking to the bottom; on *agar* a pale greyish-white streak is formed at the site of inoculation; on potato a fairly thick white film; *b*, small-sized cocci often arranged two and two, energetically mobile, and slowly growing on jelly without liquefying the medium, the growth proceeding solely along the track of the needle in the shape of a narrow stripe consisting of non-coalescing minute points of a yellow colour, while on the surface the colony is seen as

a greyish-white, circular, slightly prominent patch with somewhat fringed edges; on *agar* the coccus forms a white streak with sinuous edges; on potato a grey film with a brownish tint; *c*, very large cocci liquefying jelly as late as three weeks after inoculation, and growing along the track of the needle in the form of a sharply-defined streak of a beautiful pink colour, with a slightly elevated pink circular patch or "cap" on the surface; on *agar* the microbe forms a freely-spreading white film with a rosy tint; on potato a thick, tallow-like, pink coat with sharply-defined fringed contours. 4. That the first two species (*a* and *b*) are also met with commonly in the water of the river Dnepr, which flows through the town (*vide* Dr. Ianovsky's bacterioscopic examination of the water, published in the *Meditzinskoië Obozrenië*, Nos. 9 and 10, 1888, p. 975), while the peculiar pink micrococcus seems to occur only in snow. 5. That, generally speaking, the microbes liquefying jelly, in falling or recently fallen snow are met with invariably in far greater numbers than in snow which has been on the ground for some time; this, in fact, very often contains only such bacteria as do not liquefy gelatine. 6. That the bacteria of snow originate partly from aqueous vapours which are transformed into snow, partly and chiefly from the air, that is, they are carried away by the snow-flakes on their passage through the atmosphere." —*British Medical Journal*, December 15th, 1888.

VIII.—THE COMPARATIVE ANTISEPTIC VALUE OF VARIOUS CHEMICAL SUBSTANCES.

"At a recent meeting of the London section of the Society of Chemical Industry, Mr. C. T. Kingzett, F.I.C., F.C.S., read a paper on the above subject. The author began by expressing his belief that 'in all probability' various antiseptic chemical agents act in a similar way upon all sorts of micro-organisms, either (1) by killing the organisms directly, or (2) by causing their death indirectly by altering the composition of the medium in which they live. He considers that this view

receives support from a careful study of the chemical changes which are produced by various micro-organisms, all of which may be classed, according to Mr. Kingzett, under two heads — namely, hydration and oxidation. Moreover, he believes that the most important chemical poisons (ptomaïnes, etc.), which are produced by certain pathogenic micro-organisms, are formed by a process of hydrolysis, set up by the organisms themselves.

"Having thus stated his views as to the practical importance of studying the action of various 'antiseptics' (germicide), he proceeds to describe three series of experiments bearing on this point.

"In Series 1 the action of chlorides, nitrates, and sulphates of various metals was tried, with the general result that these salts of the metals of the alkalies and alkaline earths were found to be very deficient in antiseptic power, whilst the corresponding salts of manganese, zinc, tin, iron, lead, and aluminium have a decided value, whilst the metals, copper, and mercury, are superior to all others in this respect, mercury being better than copper.

"In Series 2 the antiseptic action of 'sanitas,' 'salufer,' and 'bactericide' (three antiseptic solutions patented by Mr. Kingzett) are compared with solutions of the more potent antiseptic metallic solution of Series 1, with results which are favourable to the new solutions. Indeed, the author asserts that his solutions, which contain peroxide of hydrogen, possess an advantage over mercuric chloride, in that they act as oxidants, which the latter does not and cannot do.

"In Series 3 a number of experiments are described in which mixed solutions were employed, some of the results being highly interesting; for example, chloroform is found by Kingzett an inferior antiseptic to chloral; boric acid he finds superior to borax, though borax is said to be superior to a mixture of borax with boric acid added to enact neutralisation; and, as a rule, acid solutions are found to be better antiseptics than alkaline ones.

"The author has tabulated his results, and proposes that his tables shall be used for purposes of comparison in case of the

introduction of any new antiseptic solutions."—*British Medical Journal*, November 24th, 1888.

N.B.—On the subject of ANTISEPTIC MEDICATION reference should be made to the valuable and practical contribution of Dr. Shingleton Smith, which appeared while these pages were in the press, entitled "Some Recent Developments of the Germ Theory," etc.

OPINIONS OF THE PRESS ON OTHER WORKS BY DR. DOBELL.

See Titles, p. 135.

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"'On Diet and Regimen in Sickness and Health.' An exposition of the hygienic questions relative to ventilation, heating, sleep, exercise, posture, bathing, regulation of the bowels, rest and change, meals, etc., in addition to the all-important subject of diet. . . . The rules seem to us very judicious. . . . The alcohol table is most ingenious. . . . . We report favourably of the idea which our author has broached, and of the manner in which he has brought it forward."—*Dublin Medical Press.*

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TITLES OF THE WORKS REFERRED TO IN THE FOREGOING REVIEWS.

1. ON THE CLASS OF MEDICAL LITERATURE MOST NEEDED IN THE PRESENT DAY. (Out of print.) Wertheimer.
2. PRACTITIONERS' AND STUDENTS' GUIDE TO PHYSICAL DIAGNOSIS (Demonstrations of Diseases in the Chest, with Coloured Plates). 12s. 6d. Churchill.
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